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TRANSACTIONS

OF THE

NINETEENTH ANNUAL MEETING

OF THE

American
Academy of Ophthalmology
and Oto-Laryngology

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HELD AT
BOSTON, MASS.
OCTOBER 19, 20, 21, 1914

The Twentieth Annual Meeting of the American Academy of
Ophthalmology and Oto-Laryngology will be held at Chicago, Ill.,
Oct. 5, 6, 7, 1915.

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RAY, J. M., Louisville, Ky.....	1914
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THE PRESIDENT'S ADDRESS

J. MORRISON RAY, M.D.
LOUISVILLE, KY.

Ladies and Gentlemen, Fellow Members of the American Academy of Ophthalmology and Oto-Laryngology: In selecting me as the president of your Society you have conferred the highest honor at your disposal, an honor of which any member might well be proud. The American Academy of Ophthalmology and Oto-Laryngology is the largest society in the world devoted to the study of the diseases of the eye, ear, nose and throat. The gentlemen who have preceded me in this chair were eminent in their special line of work. Allow me to thank you most heartily for the honor. It shall be my endeavor so to fill the position that you will not regret your selection. To obtain the confidence and recognition of one's associates and colleagues is the most pleasant experience that comes to us as professional men.

It seems wise to allow the program to furnish the material for scientific thought and discussion and permit the presiding officer to devote his efforts to a review of the work of the Academy and to some suggestions relative to its present welfare.

The two publications with which this society has been closely allied and which it has done much to promote furnish each of you with a list of important contributions to our specialty, and when combined with the Year Book and special journals, offer a most comprehensive up-to-date résumé of the year's progress, making a recapitulation of the literature of the year inadvisable at this time.

The growth of this society has demonstrated the need for such an organization. It was inaugurated at Kansas City, Mo., in April, 1896, with a membership of fifty, under the presidency of Dr. Adolf Alt, and known as the Western Ophthalmological, Otological and Laryngological Society. This number embraced men interested in these subjects either alone or combined. In many of our cities are to be found men doing work in all these branches; especially is this true in the South and West. The intention of the founders was to bring into its membership all these men. In a few years the society grew

so rapidly that it was necessary to enlarge its scope. It was pointed out that the older societies were largely made up of men from the more thickly populated eastern part of our country and that these societies were necessarily limited in membership; therefore the necessity for a virile, progressive, wide-awake organization to teach and train the large and ever-increasing body of young "would-be specialists" found throughout our country.

But our founders built better than they knew. In a few years its activities had so increased that it was thought best to extend its influence and enlarge its scope and territory. The name was accordingly changed to the American Academy of Ophthalmology and Oto-Laryngology and it was divided into two sections.

The word academy signifies a place of higher learning, an institution for training and education in any special subject, in other words, a school in which some special art is taught, or, as the encyclopedia says: "In its modern acceptance it may be defined as a society or corporate body having for its object the cultivation and promotion of literature, of science, and of art, either severally or in combination, undertaken for the pure love of these pursuits with no interested motive."

In this society we find embodied to its fullest extent this definition. The by-laws say that all men interested in these special lines of work are welcome. It was organized on liberal lines. It desires to have within its fold all worthy, aspiring, conscientious men, especially the younger ones with ambition to obtain knowledge. There is no exclusiveness as regards membership; any and all who can add one fact, clinical or pathological, will be received with open arms. If they can help us we will try to help them. The young man is the bone and sinew of every medical organization. He it is who generally has the time and enthusiasm for research work. A large percentage of the text-books now used for teaching in our medical schools were prepared by men under the age of 40. I have heard it remarked in Great Britain that all the text-books are written by young men and on the success of these the reputation of their authors in later years is largely based.

In the few years of the existence of this society we find, by a perusal of its published transactions, a strong tendency to exemplify the tenets of its name, in other words, to impress the membership with the importance of broad training and

thorough teaching in the upbuilding of the specialty. For instance, such papers as that found in the 1904 Transactions, by Dr. Edward Jackson, point out the great value of special training for the education of the ophthalmic practitioner. He emphasizes the fact that while there are more than two thousand men in this country who are devoting themselves to the practice of diseases of the eye, ear, nose and throat, the facilities for obtaining special training in these branches is meager and insufficient. The special hospitals and the postgraduate schools until recently furnished them the largest opportunity for special training. Fortunately, as a result of the influence of Dr. Jackson's teachings, several of our universities have undertaken the formal training of the specialist. The demands of the medical curriculum of the present day are so extensive that but little time can be devoted to any special line of work. Therefore I believe that until he has finished his general medical training the student should spend but little time in any special line. However, I am convinced that this special training should be given very soon after he has obtained a comprehensive insight into general medicine. For instance, the eye is so closely associated with diseases in other parts of the body that the student of this specialty necessarily obtains a certain knowledge of general medicine in the pursuit of his special training. Membership and attendance at local and county medical societies are excellent methods in the postgraduate training of the specialist in general medicine. In the 1905 volume of the Transactions, Dr. Alleman discusses the training of the undergraduate in ophthalmoscopy and wisely, I think, comes to the conclusion that much time is lost in trying to teach students the diagnosis of intra-ocular diseases. Each one of us can remember the days, weeks and, in many cases, months before we were able clearly and confidently to make out in detail the fundus oculi. I am persuaded that when we acquaint the undergraduate student with such external diseases as can be diagnosed by careful inspection, we have given for him about all the present college course can possibly give. In the 1909 Transactions Dr. Fisher advocates the teaching of ophthalmoscopy to undergraduate students, and suggests the use of the schematic eye as of great value in facilitating this idea.

In teaching students of medicine ophthalmoscopy it is important to remember the impressive remarks of Loring, "Five-sixths of the art of ophthalmoscopy are contained in a knowl-

edge of the normal eye. The rest is a series of representations which can be read almost at sight."

In the 1908 Transactions of this society will be found a number of most excellent papers dealing with the training of the undergraduate and the graduate in medicine in special lines of work. Those dealing with ophthalmology by Drs. Vail, Wood, Connor and Jackson are of especial importance to both pupil and teacher. The article by Dr. Connor on the advisability of teaching undergraduate students of medicine the practical application of the laws of ocular refraction and the method of their correction brought out a valuable discussion. His thesis was intended to meet the needs of many general practitioners of medicine and at the same time combat the rapidly extending tendency to form a new class, aspiring for professional recognition and known as optometrists or doctors of optometry. If a course in refraction and the application of glasses to the correction of the more simple errors of refraction be made a part of the undergraduate instruction in all medical colleges, I am sure that it would do a great service in the eradication of the so-called "spectacle doctor" and traveling optician, who are now so much in evidence.

The Convocation at Minneapolis, the papers presented to the Oxford Congress last year, and the recent discussion before the British Medical Association all show that we are beginning to impress the university authorities as well as the profession as a whole with the importance of ophthalmology in the curriculum of the undergraduate medical colleges.

While I have thus briefly emphasized the work of this society in promoting the training of those who desire to take up special practice, I do not for a moment wish to underestimate the valuable contributions found in the Transactions dealing with physiological, pathological, surgical, congenital and clinical facts. The reader is amazed at the large amount of valuable material found therein. And my wish is that the future will add still more to the great work, as there are many ocular conditions whose etiology and pathology is still imperfectly understood and on which more light can be thrown. As has been well said, "In science, as in art, there may be pause, but there can be no finality." Therefore the full fruition of our many hopes and ideals may probably never be witnessed by the coming generations. Perfection, though approximated, can never be attained.

The impetus given to the study of genetics and its influence on eugenics has become an important factor in our present-day

sociological propaganda. The study of heredity has already furnished many interesting problems and its promises for the future are great. It has been conclusively shown that the hereditary descent of certain conditions can be proved to follow definite predecidable formulae. In no part of the human organism can these changes be more accurately or successfully studied than in the human eye and in no organ are the evidences of transmission more often shown or the laws of heredity more invariably followed. We find in the proceedings of former meetings of this society a number of interesting observations and one very extensive study of the subject by Dr. Loeb, and I would urge the members of this society carefully to note all cases wherein the evidence of heredity is apparent, in order that the future study of this vital subject may be facilitated and valuable deductions obtained.

At the May meeting of the American Ophthalmological Society a committee, of which Dr. deSchweintz was chairman, made a report dealing with the question of granting a special degree in ophthalmology as part of an optional course in universities and medical schools of the first class. The report urged that this degree be conferred only after the applicant has had a course of two years' study and had previously obtained the degree of M.D. The special character of the degree to be granted the committee recommends should be entitled master in ophthalmology. The report was adopted by the society and a request was made that the committee appointed from this society last year, of a similar character, consider the subject and cooperate. I am glad to announce that this committee will make a report at this meeting.

This society stands for upright, professional conduct and honorable dealings with our fellows. Therefore, we deplore those acts that discredit our profession with the laity or lower our standing as reputable practitioners of an honorable calling. In commerce and trade the accumulation of money and property is commendable. The object of a profession, however, is the diffusion of knowledge and the upbuilding of mankind, hence it should never be mercenary. I feel sure that the membership of this society deprecates the growing tendency to commercialize the profession. We should make this society such that membership in it will be a valuable asset. Let it be a testimonial to the member's fitness to practice the specialty he has selected. Let it bring the fellows into close professional touch with one another. Let it be an impetus to study and an inspiration to the attainment of

high ideals. Let us be able, wide-awake workers imbued with the scientific spirit.

It has been said that ophthalmology is such a fascinating subject that there is danger that those who enter it will specialize too soon and too much. I would emphasize the statement that no one can safely and intelligently deal with the many defects and diseases of the eye without an adequate knowledge, not merely of its anatomy and physiology, but of its relationship to the whole organism.

As a result of the unfortunate condition likely to exist in foreign countries for several years to come, the responsibility for training men in special work is largely increased in this country. Therefore it seems that some immediate effort should be made to bring this about. In 1903 Dr. Ballenger offered a resolution deeming it wise for this society to cooperate with other societies in America devoted to eye, ear, nose and throat work with a view to the formation of a confederation for the consideration of scientific matters relating to these specialties. I would earnestly suggest this to be a most appropriate time for the consideration of this resolution. The opportunity now offered for this country to place itself at the forefront in all departments of scientific medicine should not be lost.

My hope is that this society will not only increase in numbers, in influence and importance as time goes by, but also in professional and public esteem.

It is pleasing to state that in all which constitutes a prosperous and successful society my report is favorable. The program as arranged is most excellent.

It was the desire of the Council to furnish a pre-session reprint, which has proved successful in similar societies, but the unlooked-for war in Europe marooned many, who were unable to furnish the material in time to have it printed and distributed.

I am sorry to inform you that for the same reason Professor Alexander, who had promised our vice-president, Dr. Ingersol, that he would be present, cannot be here.

Allow me to express on behalf of this Academy our full sense of appreciation of the intelligence and labor which our industrious secretary has devoted to the welfare of the society and his efforts to make the present meeting a success.

Allow me to thank the profession of the city of Boston for their generous arrangement for entertainment and assistance in carrying out the program of the society.

HOW SHALL WE TALK ON THE CONSERVATION OF VISION?

JAMES A. SPALDING, M.D.
PORTLAND, MAINE

Now that the National Committee on Conservation of Vision has decided on a second campaign for the coming winter, the best way in which we can prepare ourselves for successful work is to bring the whole topic before this Academy for discussion. Some of the lecturers on conservation read from manuscripts, some talk off hand, some have a good delivery, others talk too scientifically, some believe that slides are indispensable, and finally there are some who, with me, believe that the best way in which you can drive your thoughts home is to look at your audience and make them look at you. Some of my listeners have been kind enough to say that I talk well, but I take such compliments with a grain of salt, wishing I might talk better still, and never forget to make every point that I ought to make as it comes along in its turn.

If we wish to accomplish the purpose of the national committee, we must place our facts before the public in an interesting way. This is best done by being interested ourselves. We can arouse enthusiasm only by being enthusiastic ourselves. We can make people believe that the most precious possession in life is a pair of good eyes only by believing so ourselves and making evident to the people that belief. We must keep in mind, every word that we speak, to appear totally and completely interested, and enwrapped in our subject. Beyond that, also, we must be so well informed on all the aspects of conservation that we can not only speak well our own thoughts, but are prepared to answer off hand any questions that may afterwards be handed in. On one occasion thirteen questions were put to me, and I was obliged to reply without hesitation what I thought was the best electric light, the best kind of chalk for blackboards, what I thought of the goggles of to-day, what could be done for cross-eyed children, and so on.

Such being an ideal for obtaining success in talking, I come here to ask your advice as to the best ways of outlining conservation. How shall we talk on this topic so that people will

believe that we consider it the greatest one in the world so far as public health is concerned? Cancer, tuberculosis, syphilis are good topics for talk, but it is not everybody who suffers from these diseases. It is, however, of paramount importance that we should have two good eyes with which to see how to fight in the battle for existence, and that we should keep those two eyes good until the natural decay of age shall dim them beyond human skill.

In order, now, to get at your opinion of how we ought to talk on the care of the eyes, I know of no better way than to tell you what I have been doing in Maine.

When the oculists of Maine chose me a committee of one to carry on the campaign, I went home, accepted the appointment by letter to the National Committee before I went to bed, and I then proceeded to type off ten sheets or more as a beginning of a lecture. When the literature on the topic arrived, I read the dozen pamphlets, put them aside and from memory typed off fifty sheets more. On top of all that, I recalled from my practice of forty years all of the instructive injuries and interesting facts of the connection of the human body with the eye that I could. I was, as I thought, ready for a call, but when it came and I looked over my material I saw in a flash that I could never utilize it for an audience, but that I should have to go all over it and sort it out this way and that. In this way I soon found myself with notes for an audience of physicians, of nurses, of teachers, of superintendents of schools, of college students, and of societies of women. Some such arrangement appeared imperative, for what might suit one sort of audience would not at all appeal to another, because various people would look at care of the eyes from so many different points of view that each would have to be satisfied to become interested in the lecture.

I always introduce what I have to say by mentioning that the American Medical Association has asked me to speak on conservation of vision, or care of the eyes, and that although some physicians seem to think that these public appearances savor of notoriety, yet the cause is so important to the community that we are obliged to put sensitiveness aside and make the best of what the faultfinders may say. My preparation consists of a few notes, mentioning the headings of the topics which I have decided to discuss for that particular audience. For a meeting of teachers, consisting much to my surprise of more than seven

hundred in all, in a large hall, I wrote these eight words on a bit of paper: moving pictures, truants, cross-eyed children, sight testing. My only illustration was our set of test type. From those notes I spoke on the good and the bad points of moving pictures, the best way in which to make them educational, and the improvements that were imperative before they should cease to be a danger to the eyes in some respects. Such pictures should be moved mechanically and uniformly; slower, too, and the hall should be illuminated at intervals. The pictures should stay longer on view, people should not be allowed to sit too near to them, and especially they should not be compelled to look up at them at the expense of the eye muscles.

Truants were discussed as having some physical defect of which they were ashamed; their eyes might be defective and need fitting with lenses; many of them could not see blackboard examples; could not read long at a time; spelled badly from not seeing well, and were ashamed to acknowledge their defect or were not even aware of it themselves. Such children needed a friendly examination by a physician or expert and should receive encouragement.

Eyesight testing was thoroughly discussed. Children were not to be allowed to learn the letters by heart, as often happened by frequent exposures of the types, so that they could repeat them all without actually seeing them; but a proper testing required two persons, one to cover each eye separately, and the other to point at this or that letter, and cause the child to name it. Furthermore, when children seem to see perfectly with each eye, yet complain of headache or scowl habitually, they should be referred to expert oculists for a skilled examination. Cross-eyed children need attention by obtaining a perfect set of lenses, and then each eye should be daily covered from vision in order to exercise the other. At a certain age, if not relieved in these ways, an operation should be done.

When speaking to superintendents of schools I discussed the lighting of schoolrooms, by day and especially at twilight, or on cloudy days or during the passing over of heavy clouds or rains. Windows should extend to the ceilings to get all possible light. When adjoining buildings shaded a schoolroom, the new prismatic arrangement to catch the light on the highest row of windows and throw it farther into the room was recommended. Individual electric lights with a shade to keep off the heat of the light as well as too direct light were advisable on desks far

from windows. Great stress was laid on the fact that most chalk marks made on blackboards, as in copies for writing or examples in algebra or arithmetic, were not thick enough in comparison with the width of the standard letters to be seen at thirty or forty feet. I here went to the blackboard and showed how much farther off chalk marks could be seen if so standardized than if made by guess work by the children. Most of the marks left on the boards by the children the day before could be seen at only fifteen feet without eye strain, while mine were visible at forty feet.

Superintendents were also told of bad type, bad spacing of type, German and Greek type altogether too small and bad for the eyes. My idea in going into these minutiae is that superintendents might have some influence with publishers and thus obtain for the children better-printed books with corresponding decrease in eye strain. In this same direction I showed where our music charts are defective, for they should be based on the width of the lines of test type that should be seen at forty feet. As at present printed, the lines of the staff, limbs of the notes, outlines of the half and whole notes, as well as of the naturals, sharps, and flats are below the standard of test type that should be seen clearly across the average schoolroom. All of these defects produce eye strain, and it can only be remedied when the state superintendents obtain standard charts based on the average size of schoolrooms.

I have found state superintendents also interested in special schools for the short sighted, schools in which teaching shall be done more by the ear than by the eye, and with less eye strain in young myopic children. In such schools each child should be tested as to sight, fitted to proper lenses and so seated as to relieve all possible eye strain. Those of us who have been long in practice and have watched the gradual ruin with age of so many short-sighted persons view with regret the eye strain nowadays demanded of the myopic and hope that special schools may prevent this damage to another generation. Superintendents also should have weight with boards of health in keeping children out of school longer than formerly for that form of eye strain which follows the exanthemata and the mumps and tonsillitis.

Let me here emphasize the fact that I lay great stress on the advancement of conservation from talks with school super-

intendents, for these officials exercise a wise and beneficent influence throughout the nation.

Teachers in Normal Schools should be talked to in a similar direction, but more stress laid on their personal influence on the scholars. They should be told of the accidents from games, the need of care of the teeth so far as they affect the eyes; they must be precisely instructed in test-type use, as well as in the use of pencil, pen and ink, proper position at the desk in writing; they must see that the schoolroom is properly lighted on gloomy days; they should inform parents of need of lenses for their children, and be on the watch to observe that the frames of those children who wear lenses shall be kept well set on the nose, and face. They should talk of the idleness produced by too much moving picture shows, and keep track of convalescent children lest they begin to use their eyes too soon after an illness and so bring about eye strain. At this point we can recall cases from our practice and show the teacher that the eye is a part of the body, influenced by the teeth, the heavy lunch at school, the drinks, the ice creams, the indigestion so resulting, the prolonged use of the eyes and so on. We should spread the gospel that the eye is largely dependent for its functions on a good condition of the human body. We should finally urge special attention by the teacher to the safeguarding of the eyes of children in the mechanical training schools. For it is easy to see how, with tools to which they are unaccustomed, they may injure their eyes, to say nothing of instruction against the careless driving of nails or tacks, or of opening boxes and barrels.

To the members of a women's club, I should talk fully about accidents to children of all ages, going into detail of all that we have seen in our practice or read about, from pointed edges of furniture, knives, forks, needles and pins; urge the use of safety pins invariably. Tell how a napkin pinned with a common pin, being pulled off over the child's head, injured an eye. Mention orbital injuries; show how the running against the sharp edge of a door in the dark, or the bending over in the dark and hitting the knob of a chair may destroy an eye.

Tell them of injuries from baseball bats and how falling from a cycle on the forehead and injuring the orbit may destroy the sight. Show how people scratch a match toward them, from the bottom of a match box and get a chemical burn of the eye. Think up and talk of your interesting cases of injuries to the

eye, and then cap the climax by telling that you have talked much on accidents because many of them cause loss of sight in the other eye by sympathy and at this point show a slide with the picture of the optic nerves crossing from one eye to the other, or draw it on the blackboard.

To such an audience you want to go into the subject of trachoma, show the granules on the everted lid, explain how they "sandpaper" the cornea and produce blindness. Write out the word cornea, and don't let people go about talking of the corner of the eye. Write out also the words normal eye at this point and explain that it is not a nominal eye, as is often the case. Show then the pictures by Dr. Stuckey of the trachomatous blind, and from that argue of the need of keeping out trachomatous immigrants, lest all parts of America become afflicted.

Wood alcohol is another important topic for women to understand, for they can influence legislation against this horror. Tell them of its dangers to the sight from inhalation, from handling wood pencils, from drinking. Do not forget to tell them that four tablespoonfuls of wood alcohol have produced almost total loss of sight, as of late reported. The world has got to be kept up to the mark by some means or other, and women can help us. It is easy to laugh at the failures of prohibition, but there can be no doubt that the consumption of liquors should be kept down to the minimum. Some alcoholics steeped with that poison do live longer than some who do not drink at all; but what the alcoholics accomplish in this world is far below what they might have accomplished with less liquor and still farther below that accomplished by reasonable abstainers.

To women also we must urge the prevention of the ophthalmia of infants or, as in my opinion we ought to call it the sore eyes of infants. I find it best to make it plain that although all infants are liable to sore eyes, only few are actually infected, and that owing to prevention there are fewer cases than of old. The reason for mentioning a disease which none of them ever perhaps saw is that as it does occur and as they may hear of an instance, they should urge instant treatment to prevent blindness. Sore eyes in infants are a danger not only to the infant itself, but to the mother, the nurse, and the attending physician from contagion. Prevent it if possible; obtain proper treatment at once, is the gist of a talk from this point of view.

Women should also be instructed in the dangers of patent medicines for the eyes, because they contain cocain and sugar of lead and even atropin; in the dangers of hair dyes and face powders, as they too may harm the eye; and likewise in the careful use of hair tongs, which have been known to burn the eye and affect the sight permanently.

In conclusion of the topic of talks to women's clubs, I always bring up the workmen's compensation laws, which are sure to become universal; mention the dangers to the eyes of their husbands, brothers, or relations from chips of iron, steel, copper and various metals, and ask their co-operation in urging compulsory goggle-protection of the eye in dangerous trades.

When we talk to trained nurses we have to exercise a certain discretion. Many nurses are more up to date in treatment than certain physicians, but they cannot interfere with bare-faced suggestions for conservation of vision. We cannot teach a nurse to ask a physician in a lying-in room if he ever heard of an eye being injured by the forceps. But she can be taught to inquire if the forceps ever slip and harm an eye. To such a question the physician may say he never heard of such a thing, but the suggestion will make him more careful in applying the forceps.

We may not teach a nurse to say that she is now going to put nitrate of silver into the baby's eyes, but we can urge her to ask if she shall now use an antiseptic in the baby's eyes. Suggestively, then, you are to talk to nurses on the care of the eyes during etherizing lest they abrade the cornea and then go into various injuries to the eyes which they may see in families or be called on for first aid. We can teach a nurse to say of an injury of the eye, "That looks trifling to me, but better be sure, for that child may need two eyes for possibly seventy years of life and now is the time to be sure just what has happened." I am firmly of the opinion that an hour's talk on care of the eyes once a year to every trained nurse in every hospital will be of immense benefit to the state and nation. For, these capable young women go into every household sooner or later, and good, sound, plain advice to them on the care of the eyes and conservation of vision will travel far and do much good to all the people.

I have talked to groups of college students, and as the locality where I first lectured happened to be a chemical lecture room, I began at once on the dangers to the eyes from chemical

explosions, from the tumbling over of bottles when lifted from a high shelf, and the contents falling into the eyes; of the bursting of ammonia bottles, and other injuries from careless handling of chemicals. From that I passed to gonorrhea and syphilis and their dangers to the eyes. In mentioning gonorrheal ophthalmia I went into the dangers of contagion, care of the hands and cloths used in cleansing, and the danger of gonorrheal iritis. From that to syphilis and the long train of its eye diseases the step was easy. I told of the cases that I had seen, emphasized the risks of the damnable disease, and the eternal care to be taken against it in every shape.

In talking to physicians, as at county medical meetings, I begin with the dangers to the eyes from the obstetrical forceps, tell of the cases seen and reported, including evulsion of the eye, and urge care in applying these useful instruments.

I then go to the prevention of ophthalmia in infants by silver and its salts and add that some physicians who have practiced for years and never used a preventive have never yet met with a case of ophthalmia. On this I congratulate them on their good fortune. Personally I have seen many cases, every mother was delivered by a physician, and such cases must come from somewhere. Therefore I insist in my talks on the preventive treatment of infantile ophthalmia. I urge it for the sake of the child, then for the sake of the oculist who will treat the child and may suffer from the disease in his own eye, next for the sake of the nurse, who may lose an eye in treating a case of ophthalmia, and I tell them of three nurses in Maine now wearing an artificial eye from an eye lost from infantile ophthalmia; and finally I urge prevention for the sake of the physician himself. For in a case of blindness some shrewd lawyer may get up a malpractice suit against you. What answer are you going to make when that man asks you: "Doctor, did you use any of the necessary scientific preventives against the ophthalmia of infants which has made this child blind?" How are you to satisfy a jury that you have not been guilty of negligence? How can any specialist summoned by you as an expert in your favor give you the least honest assistance?

Much can be made of the treatment of ordinary eye diseases when talking to physicians. Show them how to apply iodine to an ulcer of the cornea. Make plain the first aid in injuries. Show country physicians the dangers to weeping eye from a barb of oats or barley. Tell them of sympathetic ophthalmia,

and go into the need of careful testing for lenses and the great benefits of lenses in headaches and nervous affections.

As you see, the aspects of conservation are enormous and not to be exhausted in a dozen lectures to different audiences. I have mentioned a few with a desire of bringing out more. Little as we think we know, the people know less and it is our duty to inform them. You will find them asking odd questions. "How much wood alcohol will it take to make anybody blind?" "What do you think of moving pictures?" "What is astigmatism?" "What do you think of opticians?" "What do you think of the new goggles?" To the last question my answer is: "These goggles are not new at all. I have seen them on the portrait of a Spanish cardinal who was painted as far back as 1595 and that portrait is still in the Prado in Madrid. Some people called these goggles "Chardins," from a portrait in the Louvre painted about 1760, but that was many years later than the painting of the cardinal. They ought to be called "Cardinals," not "Chardins;" besides, if you are careless in wiping the lenses they will easily revolve out of their proper setting.

Some one may here ask, "Don't you ever talk of refraction?" I say positively, "No, unless somebody asks me." Then I show a normal eye, one larger and one smaller, side by side, and explain the difference. And if they pester me with, "What is this astigmatism that people talk so much about?" I say that the astigmatic eye is flat one way and round the other and by putting on a round lens to the flat side, the eye becomes all round and can see clearly. Refraction opens too wide a field for ordinary audiences and soon their eyes close into absence of comprehension of the lecturer.

Do you ever mention the opticians? Never. If asked, I say that these men have never been examined to see if they know anything about the eyes or the eyesight. If they then reply that such men have been legalized by the state, I reply that they paid the lobby to get exempt from examinations, and were registered without examination. The people are now paying such unexamined, unqualified men more for lenses than they ever paid before for the skilled advice of a trained and examined oculist and lenses added.

The most puzzling question in this campaign is how to get invited to lecture. Circulars are useless. I once thought of sending out a syllabus. I look now for suggestions on this point from you. My latest move was to make a journey of more

than 500 miles to attend a meeting of delegates from all the women's clubs in Maine, to outline our lectures and to urge them to invite my lecturers and myself. Owing to this journey, I have received already several invitations. If people ask our price, we may ask for expenses only. Owing to the extra fares of a man to manage the slides, I have always dispensed with them, because money is hard to raise in such campaigns. I find that a few illustrations handed around and the use of the test types, music charts, and blackboard interest most audiences.

In conclusion, let me regret exceedingly to say that some physicians have refused to speak on the care of the eyes because it is unethical and self-advertising. Yet, fellow-members, it is all right to advertise as an eye and ear surgeon on the staff of a hospital, or to advertise your private hospital, or to cause or permit to be inserted in the newspapers items to the effect that John Smith is undergoing a serious operation for his sight in Peter Robinson's private eye, ear, nose and throat hospital. That is ethical enough and so is it ethical to flaunt on letter heads and in the papers, professorships in medical schools. All this is ethical because they all do it or would do it if they had the chance. This is for you to consider. My opinion is that every member of this Academy who comes to the relief of the people by lecturing on conservation of vision is a benefactor of the state. If it is contrary to the code of ethics to teach the people how to safeguard their eyesight, then on that code I am going to turn my back, and take the consequences, which I consider trifling indeed in comparison with the good that I may be able to do for the great cause with my voice and my example. If I have to be the only man in Maine to carry on the approaching campaign, I will gladly take it all on my own shoulders and pay all the expenses. I am, however, grateful for a few excellent men who have come to my assistance. To them my thanks, and to you more thanks for your attention.

DISCUSSION

DR. F. PARK LEWIS, Buffalo, N. Y: A subject of great importance, and one that has hardly been touched upon, is the attitude of the general medical profession toward sight saving. If we will look over a few hundred of our records at random, we will be astonished at the large number of cases in which the vision in one or both eyes is below normal. Many of these show evidence of old lesions that prompt treatment of the proper kind would have prevented, but in which sight had been diminished or lost because the right thing was not done at the right time. This, in many cases, is due to a failure on the part of the patient himself to recognize the importance of threatening premonitory symptoms.

It is frequently due to an unwarrantably long period of watchful waiting on the part of the physician who has been in charge of the case.

No one expects the average general practitioner to be skilled in all of the specialties, but he should be able to recognize the oncoming of conditions anywhere in the human system, which unheeded and uncorrected will menace the integrity of an important organ or of life itself. The practical knowledge necessary to prevent the loss of an eye is not difficult to obtain. There are not many serious diseases the existence of which may not be shown by three simple tests: First, by a diminution of sight; second, by the appearance of the eyes; third, by pain.

An accurate diagnosis is not always to be expected, but the physician should always be able to determine whether the patient should have skilled special treatment or not.

Every doctor should have suitably placed in his office a visual test card. He should know how to determine and to record the degree of sight in each eye separately, and roughly to take the visual field with a white card or candle. He should know what a diminution in the field of vision means. He should be familiar with the more prominent symptoms of eye strain, and should be able to intelligently advise his patient of the importance of an accurate refractive test, and of the dangers of an inaccurate one. He should know how to use oblique illumination, and should recognize all important changes visible to the naked eye. He must know which of the conjunctival and corneal troubles are dangerous to sight, and which he may safely treat. He must not tell the parents of squinting babies that they should wait until the child is ten or twelve years old before anything is done for the correction of the squint. He must distinguish between cataract and glioma, and between iritis and glaucoma. He must be able to intelligently give first aid to an injured eye. In a word, the general and the special practitioner must have clearly defined limitations as to the work which each undertakes to do, and these are based upon at least an approximately correct diagnosis.

If every doctor were to limit himself to the treatment of those things which he knows he is safe in treating, and either getting competent advice or refusing to treat those cases which he does not understand, there would be many eyes saved which are now lost. It is only fair to the patient to ask that this be done. It is equally only fair to the doctor that he should be given an opportunity to acquaint himself with the simple methods of procedure that would give him this assurance. The younger college men have generally acquired this training, but there are many of the older generation to whom everything pertaining to the eye is a sealed book.

Would it be too much to ask that each county society give some time during the season, one evening, to a demonstration of those things about the eye that every doctor ought to know? If this were done, the saving of eyes that are now lost because someone did not know just what to do at a critical time would be enormous.

DR. HIRAM WOODS, Baltimore: In Maryland we have been trying to work along the line of conservation, and a number of the points Dr. Spaulding has raised have been brought to our attention. The great difficulty is to interest the general profession. It is hard to get conservative literature seriously read by the general profession, whose attitude toward this question of conservation of vision seems one of indifference. It is difficult, even when men are interested, to train them to present the subject properly. Too large a field is usually taken in one lecture. I never accomplished as much in any effort for conservation as I did in addressing an audience of about seventy-five pregnant women,

under the auspices of the Y. W. C. A., in the squalid quarters of Baltimore. I talked about the dangers to the babies under certain conditions, postpartum care of the eyes, etc.—but one subject. I know I was instrumental in turning many of them from midwives to hospitals.

We should call attention to the need of schools for children with defective sight. We have met children with corneal or lens opacities and incurable visual defects. In most states, my own included, there is no provision for the education of these children except the blind schools. Either they cannot see the print in the usual schoolbook, or eye strain upsets the results of such work as they do. The eye is still further damaged. There should be a modified curriculum and special books for these children with defective vision.

In this whole conservation business you have first to interest the general profession. The American Medical Association has done two things. It has studied out the usual cause of blindness or injury to sight, and has provided simple, instructive literature which can be obtained almost for the asking. We have enough literature to swamp us; but what are we to do with it? We will have to impress upon the general profession its duty in this matter. We have to prove ourselves unselfish, and if we can do that we may simplify the matter.

DR. EDWARD JACKSON, Denver: Dr. Spaulding spoke of the getting ready to teach people about the conservation of vision. Such teaching is important enough to justify all that he did. No preparation can be thoroughly efficient unless it extends beyond a study of the particular facts the speaker expects to present. He must be able to answer questions that arise in the minds of his hearers. He must even be on the alert to see what is passing in their minds and to meet it before a question is put. The reading of a written lecture will not serve the purpose. The speaker should look at the audience, as the audience will look at the speaker. This will increase the immediate attention and the permanent effect produced.

Every autumn I give to the freshman class at the University of Colorado a lecture on the manner of using their eyes. Even high-school students could have such instruction to great advantage. Not only would they themselves profit by it, they would go out as missionaries in the matter of taking care of vision. One essential point for students is the matter of saving the strain of accommodation by distance.

One of the most important subjects that should be alluded to before every audience addressed is the matter of illumination. It is not only important in the matter of original planning and building of windows that school directors may control. It is equally important in the management of shades and lamps, and the manner in which the student holds his book with reference to the light. It is important to the workman in the shop to use the light furnished him to the best advantage. One of the greatest evils we have to deal with in improving the conditions of our laborers is the very poor arrangement made for the use of natural daylight. The use of prismatic glass expands the area of daylight, but the general problem must be solved better than prismatic glass solves it. We must have the light thrown on the ceiling to get the diffused light, which is the best illumination.

DR. E. E. HOLT, Portland, Me.: About thirty years ago I began to give talks on the eyes to schools, teachers, and students at college with a good deal of enthusiasm. So far as getting into actual practice what I endeavored to teach the results were discouraging. There has been so much literature of a popular character relative to the functions of the human body given to the public since that time that the results of

such an endeavor should be more encouraging now than they were then. As a teacher, especially in penmanship, I found with certain pupils it was impossible to get them to sit properly, especially to keep their head up, so I had head rests made and when one of these was screwed on to the desk it would prevent a pupil from getting his head so close to his work. I was thus able to arrest advancing myopia. We should appreciate Dr. Spaulding's paper and do all we can to carry on the line of work he has suggested for the benefit of future generations. There is certainly need enough of it. I recall the case of a pupil attending school not twenty-five miles from Boston—excellent in everything but lighting. All the pupils had to see to study with was the old-fashioned gas burner, so high up on a chandelier that an ordinary-sized girl could not reach it to turn the gas on. I recommended a droplight with opaque shade. The principal said no one had ever complained of the light from this flickering gas burner before. There are many things which need to be remedied and we should assist in awakening an interest in them from the proper authority, namely, that great force known as public opinion.

DR. JAMES A. SPALDING (closing discussion): In lectures before a medical society composed largely of country physicians, I talked on common diseases of the eyes and simple remedies, and from that proceeded to accidents to the eyes, of ordinary occurrence, with suggestions for first aid. It is also well to show how to turn over the upper lid, and to remove small foreign bodies from the upper fornix and from the cornea. The bad effects of barbed seeds (wheat, oats, rye, etc.) on eyes affected with tear passage stoppage is worth mentioning. I mention ulcers of the cornea, especially, and show with a cotton tipped probe precisely how to apply to their abraded surface, a minute portion of tincture of iodine or carbolic acid.

Lantern slides are useful in lecturing, but people will not pay for them. In other words, to carry slides out of town, you have to pay the expenses of an operator, and so far I have found it difficult even to get my own expenses paid for any great distance.

Those who are interested in artificial lighting of dark corners of school-rooms should consult the *Wochenschrift für Therapie und Hygiene des Auges*, April 30, 1914, where they will find a sketch of a portable electric lamp which not only throws good light on the scholar's desk, but by its doubly folded shade keeps the heat from the head and face (illustrates on blackboard).

DR. NELSON A. BLACK, Milwaukee, Wis.: The comparatively recent educational propaganda on the conservation of vision and the still more recent "safety first" movement have created an increasing interest in the question of the protection of the eyes in various professions, trades and industries in which the eyes are subjected to heat and glare.

Investigation of the properties of a number of the tinted glasses on the market, by spectroscopic analysis, has seemed to indicate conclusively that some of these heretofore reputed to possess various desirable qualities are actually of but little more value as a protective measure, for the use intended, than clear glass.

Until very recently, in the majority of instances in case of need for protection against heat and intense light, an individual has selected a glass which has met his personal preference, this glass being accepted for use generally in the particular plant in which the individual was employed, with no effort having been made to determine the dangers to be guarded against or the absorptive properties of the glass selected.

It has not been satisfactorily proved what portion or portions of the spectrum of radiant energy is injurious. Research, however, has progressed sufficiently far to indicate that under certain conditions the invisible ultra-violet rays do produce serious and occasionally lasting injury. As this portion of the spectrum is absolutely of no assistance in the visual act, it should be eliminated.

The infra-red rays, according to some investigators, are a source of danger. As they are of no aid to vision, they should be eliminated.

That the blue and violet rays of the visible spectrum, and probably the ultra-violet rays, are the potent factors in the production of glare, is the now generally accepted view. At least their elimination in bright light, with practically no reduction in the intensity, relieves the ocular discomfort.

In certain industries the intensity of the incident light must be reduced, in addition to eliminating the injurious rays, because of its effect in diminishing visual acuity.

In view of the above, it would seem proper that a thorough investigation be made of the danger to individuals employed in industries where the eyes are subjected to the harmful effects of intense light, together with the study of the means of protection.

I therefore move, Mr. President, that a committee be appointed from this Society to undertake such investigation.

The motion was seconded and carried.

THE OCULAR SYMPTOMS OF BRAIN ABSCESS AND SINUS THROMBOSIS OF OTITIC ORIGIN

WITH REPORT OF A CASE

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It is conceded by most authorities that difficulty is encountered with the certain localization of brain abscess and sinus thrombosis, especially in complicated cases.

This was forcibly impressed on me, when after consulting many writers, I erred in the diagnosis of a complication in a case of otitic origin, which ended fatally. Although the case was unique and quite out of the ordinary, I am led to believe, after a painstaking search of the literature on the subject, that many of the writers along this line have accepted the work of a few, instead of holding necropsies whenever possible on the cases which ended fatally and reporting their findings.

It is not my intention to treat the subject of ocular and orbital symptoms in brain abscess and sinus thrombosis in an exhaustive manner, but merely to bring to your notice the principal ones, with the hope that the discussion which follows will assist in establishing the differential diagnosis and more certain localization of brain lesions complicating aural disease.

Absence of ocular symptoms does not justify the exclusion of intracranial involvement, but when considered in conjunction with other symptoms, is a valuable guide to a timely operation. Choked disk and optic neuritis, which are by some used interchangeably, are the earliest and most important ophthalmic evidences of intracranial involvement. There is as yet no universally accepted theory as to the nature of the origin of choked disk and optic neuritis. It is still unsettled whether the cause is by engorgement or by inflammation.

The principal differences between optic neuritis and choked disk, according to A. Adam in his "Ophthalmoscopic Diagnosis," are as follows:

1. In choked disk the veins are greatly distended and tortuous, while the arteries are contracted and show broad reflex stripes. In optic neuritis the arteries are nearly normal, the veins being overfilled.

2. The optic nerve head in choked disk forms a marked elevation, grayish or reddish gray in color. It cannot be demarcated from the surrounding retina which is likewise cloudy and gray. The disk appears striated and the tortuous vessels dip beneath it in places. In optic neuritis the nerve head is but slightly elevated.

3. Vision in choked disk may be normal for a time, while in optic neuritis it is much impaired.

4. The field of vision in choked disk may be normal or show concentric contraction. Central scotoma is present in optic neuritis.

Schmidt and Manz base their engorgement theory on the anatomical arrangement of the optic nerve, the three sheaths of which are continuous with and correspond to the brain coverings. The pial sheath is closely attached to the nerve, while the dural covering is much thicker and is separated from it by the intervaginal space, which contains the delicate arachnoid membrane floating in the cerebrospinal fluid.

The optic nerve is thinnest at its entrance into the sclera, where the nerve fibers lose their medulla and where the nerve becomes constricted in its passage through the lamina cribrosa. The intervaginal space ends by a culdesac within the sclera; and the engorgement advocates contend that increased intracranial pressure will project the cerebrospinal fluid into the intervaginal space with sufficient pressure to constrict and strangulate the nerve and vessels, and that papilledema is the result.

The men who maintain the engorgement theory claim to confirm it by saying that decompression of the skull causes rapid subsidence, in many cases, of choked disk and optic neuritis. The engorgement theory is questioned by Hughlings Jackson, Gowers, Leder, Elshnig and others, who say that choked disk and optic neuritis are inflammatory in character and that the cause is from the irritation produced by metabolic substances projected into the cerebrospinal fluid of the intervaginal space from the brain.

A recent theory¹ evolved is that increased intracranial pressure and inflammatory changes in the cerebrospinal lymph, cause blockage of perivascular lymph tracts of the central vessels and produce a stasis in egress of intra-ocular fluids, all of which soon leads to inflammatory phenomena.

1. American Encyclopedia of Ophthalmology by Wood.

It is further contended that the increased pressure within the subvaginal spaces does not produce stasis through compression of the lumen of the central vessels. The same authority says: "We know that the ophthalmoscopic differences are artificial, and that cases associated with high intracranial pressure may present only a slight disk change or none at all, while meningitis, pyemia and intoxicants are associated with descending neuritis, and without other evidence of increased intracranial pressure, may present the typical appearance of choked disk. So that we no longer attempt to make an ophthalmoscopic and etiologic diagnosis between 'descending neuritis' and 'choked disk.'"

The pyogenic process² ending in brain abscess, particularly when due to ear troubles, shows itself in three stages. They are:

1. The inflammatory or febrile stage, when the patch affected is in the stage of red softening and the symptoms are those of irritation, which lasts, as a rule, a few days, but at times is prolonged into a week, and usually follows the sudden cessation of aural discharge, the general symptoms being influenzal in character. There is as yet no optic neuritis, and the pupils react to light and accommodation.

2. The manifest or early purulent stage is one of pressure, in which the symptoms of the first stage have remitted, and slowing and lowering of the mental and physical functions are prominently noted. Optic neuritis has developed and advances rapidly and is perhaps more apparent on the diseased side. Choked disk is more often seen in cerebellar abscess than optic neuritis. The pupils are sluggish or altogether inactive.

3. The terminal toxemic or paralytic stage is one of pressure with great toxemia, and the deepening coma and general convulsions betoken an early fatal issue. Optic neuritis may be intense and the pupils are quite insensitive to light and are either contracted or dilated, and unequally so. The state of the pupils helps one to locate the side on which the abscess is situated. If they are unequal, the probability is that the abscess will be found on the side of the more widely dilated pupil. If both pupils are widely dilated the abscess is usually quite large. As the pupils become sluggish or immobile a fatal termination may be expected. Contracted pupils, with insensitiveness to light, have been seen in a few cases and this condition indicates that the disease is in an advanced stage and that the pus will

2. John Wyllie, M.D.

be green and offensive. Photophobia and diplopia are occasional symptoms.

Optic neuritis requires time for its development, and it does not appear inside of ten days. Optic neuritis may be progressing when other signs and symptoms of brain abscess are in abeyance. The absence of optic neuritis does not negative cerebral abscess, and its presence does not by any means make its diagnosis certain. Its value as a symptom is that if present on only one side, or if more marked on one side, that side is probably the site of the lesion. The rule is not absolute, as Uhthoff has noted optic neuritis on the opposite side from the abscess.

When the abscess is situated beneath the tentorium, choked disk is more frequent and severe than when it is located in the cerebrum. Uhthoff mentions that unilateral neuritis has been observed on the side of the healthy ear. This must be unusually rare, as Uhthoff is the only one who speaks of it, as far as I know. The case which I will describe later showed optic neuritis on the opposite side from the abscess.

Pathologic eye changes occur in about two-thirds of the cases of sinus thrombosis of otitic origin. Involvement of the lateral sinus may show choked disk or optic neuritis on the affected side, or bilaterally. If there is an extension to the cavernous sinus, the eyeball on the diseased side will be chemosed, and proptosed, the pupil dilated and stabile. The cornea being hazy and dry makes ophthalmoscopic examination difficult or impossible, but optic neuritis is likely to be present and the lids of the affected side become edematous. The remaining cranial sinuses are not usually infected from disease of the ear, except as an extension from the lateral sinus.

CASE REPORT.—Mrs. H., aged 28 years, was seen in consultation with Dr. Blair, the family physician, May 9, 1910. Purulent and foul discharge from left ear since childhood. Left ear contained a small quantity of fetid pus. Membrana tympani had disappeared. Probe detected bare bone in roof of middle ear. Hearing dull on left side. On right side hearing was normal. The left mastoid slightly swollen. Symptoms: severe and constant pain on left side of head for past three days; mastoid tender on percussion; temperature 99.4 F., pulse 104. Operation May 10. Slight necrosis. A sinus was traced through the tegmen antri and disclosed an extradural abscess, which was evacuated, and on probing gently there seemed to be a communication with the lateral sinus region. The lateral sinus was exposed, a perisinous abscess was encountered and the pus removed. The lateral sinus was opened, but was not thrombotic.

After the operation the patient was comfortable, and the temperature ranged from 99 to 100 F. until May 14, when it suddenly rose to 103. Pulse 134, nausea, chill and pain in head and left side of neck. At operation exposure of lateral sinus disclosed nothing. The jugular vein was uncovered and seen to be thrombosed. The phlebitic vein was excised. The patient got along well and was discharged from the hospital June 4, 1910. On June 13, 1910, the patient complained of right-sided headache and nervousness. Pulse 120, temperature 100 F. The right drum and mastoid were normal. Left side of head had nearly healed, and was healthy. June 13 there was irritability alternating with dullness. The vision of the left eye was complained of and ophthalmoscopic examination revealed choked disk. On the following day the eye was chemotic, patient very inattentive and showed slowness of thought. June 15 the left eye was proptosed, lids edematous and media hazy, left pupil dilated and stationary. Right eye normal. June 16 the nasal sinuses were examined, and found to be normal. Temperature 99 F., pulse 98. The left eye was greatly proptosed, lids quite edematous, cornea dry and hazy. Fundus of right eye showed slight optic neuritis. Patient very dull and apathetic. The head was trephined on left side, $1\frac{1}{4}$ inches above the meatus. The dura bulged, but was normal in color. It was incised vertically and the brain probed with a narrow bladed knife in various directions to a considerable depth, with the escape of a small quantity of cerebrospinal fluid. No pus was found. The patient grew gradually worse and died on June 21.

The temperature during the interval ranged between 98 and 100 F.; pulse between 82 and 108. Two days before death there was beginning left hemiplegia; the right pupil was dilated and fixed. It was at this stage that a right cerebral abscess was suspected, but the patient was too far gone for an operation.

Necropsy: The left side of the brain showed no signs of the trouble. The cavernous sinus was examined carefully and it was normal. A right temporosphenoidal abscess with pus, greenish in color, and with fetid odor was encountered. The abscess was about the size of a hen's egg. The left optic nerve and left optic tract showed inflammatory signs. The optic neuritis must have been caused by extension from the primary focus in the brain. The abscess was metastatic, as the right ear was normal.

This case shows the difficulty in localizing a brain abscess in a complicated case, and was unique in that the left eye signs simulated cavernous-sinus thrombosis.

DISCUSSION

DR. EMIL MAYER, New York: As to the interchangeability of the terms "choked disk" and "optic neuritis," the late Dr. Emil Gruening pointed out that this is erroneous. Choked disk is always the visible ophthalmoscopic expression of increased intracranial pressure, whereas, when optic neuritis is present there is never any choked disk.

In answer to the query as to how choked disk is to be explained when the disk is so far removed from the diseased area, Haskins states that they are true disturbances of the vasomotor system and that the sympathetic nerve, whose delicate fibers are more exposed to inflammations of the ear than any other nerve, is the chief cause of ocular disturbance in complicated ear inflammations.

While the occurrence of pathologic eye changes in sinus thrombosis of otitic origin is high, it seems to be a less frequent occurrence in brain abscess. Of twenty-one cases of brain abscess Dench reports ophthalmoscopic changes in but six patients.

An important ocular manifestation in abscess of the posterior fossa is nystagmus. This must naturally be differentiated from that of labyrinthine origin. In the former the motions are very wide, are of great intensity, and the quick component is directed to the affected side. It also has a tendency to become more marked as the disease advances.

Of important differential diagnostic significance is sixth nerve paralysis seen frequently in cerebellar abscess.

In the case of Jobson, the temporal lobe abscess on the right side was undoubtedly of metastatic origin, making it all the more difficult to localize; while that of the left side was by direct extension through the tegmen.

Of great importance in every complicated ear case is the exact state of the vestibular apparatus. The author does not state whether the labyrinth responded to the turning and caloric tests or not.

The focal symptoms presented, constant localized headache first over the left side of the head and later over the right, are of value in fixing the location of the abscess, while the eye symptoms are misleading.

Another aid in localization was the left hemiplegia with dilatation and fixation of the right pupil, but the patient was too far gone for an operation.

Even if the patient is moribund there is a fighting chance and we may well consider operation as a faint hope.

Bacteriologic data are also lacking. It would be interesting to know (since the right temporal lobe abscess was probably of metastatic origin and not due to direct extension) whether the organism in the brain abscess and that in the blood were identical.

The early diagnosis of brain abscess in the initial or latent stage is wrought with many difficulties, and often impossible; in the terminal stage it is easy, but by this time vast inroads have been made on the meninges and the prognosis for recovery after operation is very grave.

In the latent stage of temporosphenoidal abscess the clinical diagnosis is only accidentally made, as in the performance of the radical operation. Here operation is most successful.

In the manifest stage the symptoms are produced by destruction of brain substance or by invasion of neighboring nerve tracts, giving rise to such focal symptoms as convulsions, facial paralysis with paralysis of the extremities of the opposite side. This last combined paralysis is particularly indicative of involvement of the internal capsule.

In right-handed individuals, temporal lobe abscess of the left side leads to interference with the speech center, resulting in aphasia of various types, such as amnesic aphasia, paraphasia and in rare cases to optic aphasia (word-deafness).

An important symptom in temporosphenoidal abscess is paralysis of the trochlear nerve. This occurs on the side of the lesion and on close examination the patient is found to be unable to turn the eye downward and outward.

DR. JOS. C. BECK, Chicago: The cause of optic neuritis, or choked disk, I think has been proved to be a mechanical impediment. Dail of Prague at the Russian Medical Congress showed the way the veins enter the optic nerve sheath. This fact led me to a study of the relation of the spinal fluid, or rather, the result we get from spinal puncture associated with choked disk. I have never seen a case of intracranial trouble in which the spinal puncture came with a marked spurt, that did not also show a choked disk. Conversely, in the absence of the choked disk the spurt of the spinal puncture has not occurred. I have not heard in this paper the result of spinal puncture. Drs. Crowe of Baltimore and Beck of Vienna have shown the effect of vascular pressure on choked disk and have proved Dail's contention, by pressure on the internal jugular vein. It is a test applied in sinus thrombosis.

DR. C. F. CORR, Buffalo: You all know that sometimes, indeed quite often, the symptoms of brain abscess are totally absent. I have seen a boy of 12, who was operated on and—died the same night of cerebellar abscess. No connection with the ear could be found; no symptoms. Another patient, a girl of 21, who had a discharge from the external meatus, having had external ear trouble for years, was found to have had brain abscess for weeks, emptying through the external meatus. A large cerebellar abscess without any symptoms and brain abscess on the opposite side were found post mortem. The only symptom she had was subnormal temperature. With regard to sinus thrombosis, we may have very extensive thrombosis without symptoms of any kind.

DR. G. W. MCKENZIE, Philadelphia: With regard to choking of the disk, it has been pointed out by Koerner and others that there may occur slight choking together with distention and increased tortuosity of the veins in simple mastoiditis without brain complication, which clear up promptly after a simple mastoid operation.

Nystagmus is an important symptom which usually indicates trouble in the semicircular canals. Again, we may find nystagmus resulting from brain complications, especially if the lesion is in the region or the cerebellopontine angle. In the early stages the nystagmus is toward the affected side, in the late stage to the opposite side. Instead of nystagmus we may find a conjugate deviation of the eyes, especially in lesions occurring in the posterior fossa, less frequently from a lesion in the region of the angular gyrus, either an abscess or meningitis. Ordinarily it is much easier to make the correct diagnosis post mortem than ante mortem.

DR. HARRY S. GRADLE, Chicago: Let me emphasize again the examination of the blind spot. The divisions are so small that they may not be detected at ordinary distance, but if examined with my tangent scale at one and a half to two meters, we will be able to detect minute defects in this area. We can trace the course of the vessels as they leave the disk, and any disease of these vessels will manifest itself by a projection of the vessels along the blind spot.

HYOSCIN AND MORPHIN AS A PRELIMINARY TO LOCAL ANESTHETICS

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The problems we have to meet in operations of the ear, nose and throat are to banish pain (physical) and the emotion of apprehension, or fear (psychic).

The otolaryngologists have long been adepts in local anesthesia, but we have been negligent in blocking the baneful influences due to fear. This state of acute shock is frequently observed, which varies from mild symptoms of syncope to profound exhaustion, even to a fatal termination, during a supposedly safe and simple operation.

These fatal cases are frequently attributed to a lymphoid state, cocain poisoning, etc., whereas, if the baneful influence of psychical shock had been eliminated, there would have probably been no ill effects.

We are much indebted to Crile for his studies on causes and treatment of shock. He found that there were histological changes in the brain, liver and suprarenal glands.

Shock may be produced by diverse causes, such as trauma, hemorrhage, fear, worry, infection, excessive muscular exertion, starvation and insomnia.

The causes that immediately concern us are those that produce the baneful effects of the operative trauma and the emotion of fear and worry, preceding, during, and after the operative procedure. Shock due to the other causes is not germane to this paper.

The stimulation of the nerve ceptors, both of the contact ceptor on the surface and of the special senses, causes a discharge of energy which, if protracted enough, produces the condition called exhaustion, or shock. The nociceptors are most abundant in those parts of the body which, in the course of evolution, have most frequently been subjected to injuring contacts with the environment. There are specific nociceptors in the ear, nose, and throat.

Fear is most powerful in its effects on the organism. Fear, accompanied by pain, may rapidly drain the dischargeable nerve energy to the condition of shock.

The lack of faith, either justifiable or unjustifiable, on the part of the individual, in his own ability to protect himself against real or fancied hostile environmental elements, is accountable for a large part of the apprehension. We know that phlegmatic individuals are better subjects for surgical procedures than the nervous, therefore our aim should be to make the nervous type extremely phlegmatic.

The prevention of shock directly concerns us. To accomplish this, the consumption of nervous energy should be prevented, excluding all harmful physical and psychical stimuli from reaching the brain, which is done by blocking with the local anesthetic the field of operation and blocking the special senses with general narcotics. Hyoscin depresses the psychic and motor centers. This augments morphin in blocking off the special senses and reflexes. Morphin directly prevents shock (Crile¹).

APPLICATION

The class of cases in which I have used hyoscin and morphin have all been good surgical risks. The worst cases were a few alcoholics and those with slight evidences of chronic toxemia. They have been the usual run of cases that come under the surgical observation of an otolaryngologist, from submucous resection to sinus disease, ossiculectomy and malignancy of the larynx. I have never used the hyoscin and morphin in cases of extremes of age. There has been no alteration of diet; the drug has been usually given about two hours after luncheon. A state of mental quiet, where the individual has to make an effort to keep awake, with the mind free from apprehension and fear, and feeling happy and dreamy, is the ideal for which we strive. There may be a mild delirium, from which the patient will momentarily awake if spoken to loudly and will do whatever is asked. In reaching this stage there are no hard and fast rules for dosage or time.

I start with hyoscin hydrobromid, $\frac{1}{100}$ gr., and morphin, $\frac{1}{8}$ gr., given hypodermatically, after which I place the patient in quiet surroundings and under close observation. The effects of the hyoscin are usually more pronounced than those of the morphin, the pupils dilating with some flushing of the skin and a dreamy and happy expression. After thirty or forty minutes, if the patient still shows signs of apprehension or fear, I give

1. The author has largely drawn upon Crile's work, "Anoci-Association," to whom he wishes to make grateful acknowledgements.

from $\frac{1}{200}$ to $\frac{1}{100}$ gr. more of hyoscin and from $\frac{1}{16}$ to $\frac{1}{8}$ gr. more of morphin, but I vary the two drugs if the patient shows the effect of one more than the other. It may take from forty minutes to two hours to get the patient quite indifferent and in the state required for the particular operation. Naturally, a submucous resection will not require as deep an anesthesia as a case of sinusitis.

It takes considerable experience to get just the proper narcosis. If the patient is insufficiently under, the harmful impressions conveyed to the special senses of instruments, blood, and operative procedure will show in nausea, syncope, etc.; if too far under, the patient will go into deep sleep, which is unnecessary. The field of operation is anesthetized as usual.

Personally, I have ceased using cocain; not that I have had a case of poisoning, but its exhilarating effect in its first stages is not wanted and the after depression is sometimes quite distressing to the patient. I use alypin in the same strengths as in the case of cocain with the same anesthetic effect. When an anesthetic is to be used hypodermatically, novocain or urea and quinin hypochlorid is preferred.

ADVANTAGES

All shock is avoided. The patients awake from the effects of the hyoscin and morphin usually without any memory of the operation, or anything remembered is not of an unpleasant nature. For instance, sometimes a patient will have the remembrance of some hard pulling or of having heard the breaking of a bone, but meaning nothing to the patient.

Much more extensive work can be done than under local anesthesia alone and with less ill effects than with a general anesthesia. I have done such operations as the Killian, double sinusitis per nasam, removal of large sequestra of lower jaw, repair and replacement of all of the facial bones, due to a horse kick, ossiculectomy, etc., under this method.

DISADVANTAGES

It takes much time and experience. The patient should be under your personal supervision from shortly after the first dose until an hour or so after the operation is over, which means that you must give the patient your attention from three to five hours.

Its effect differs widely, due to idiosyncrasy. I have had one failure in the case of a hysterical girl, who, when the opera-

tion was half over, broke out with tears and laughter and was quite noisy for several hours.

It is unsuitable for use in clinics and most of the hospitals, unless the surgeon or some one specially trained has the time to remain with the patient. The ordinary trained nurse does not appreciate the situation sufficiently to be left in charge, as the patient is not accountable for his actions.

Commercial hyoscin is not all alike, therefore care must be used to obtain a superior product, and when once familiar with its action, stick to that one manufacturer. I use Merck's.

Two of my cases showed a marked fall of blood pressure when sitting up, one, an alcoholic, had $\frac{3}{200}$ gr. of hyoscin and $\frac{3}{16}$ gr. of morphin; the other case, one of arterial sclerosis, had $\frac{1}{50}$ gr. of hyoscin and $\frac{1}{4}$ gr. of morphin.

The pulse immediately picked up after the operation when the patients were put on their backs.

I might add that most of the patients were operated on in a sitting position, which is not so favorable as when done with the patient on his back.

Another case, one of laryngofissure, was that of a patient who was awake and mentally bright after two doses of $\frac{1}{100}$ gr. of hyoscin and $\frac{1}{8}$ gr. of morphin, requiring a third dose, the third dose making a total of $\frac{3}{100}$ gr. of hyoscin and $\frac{3}{8}$ gr. of morphin.

Another case, one of frontal sinusitis, required three doses to produce indifference: at 2:20 p. m., $\frac{1}{100}$ gr. of hyoscin and $\frac{1}{8}$ gr. of morphin; at 2:50 p. m., $\frac{1}{100}$ gr. of hyoscin and $\frac{1}{8}$ gr. of morphin, and at 3:30 p. m., $\frac{1}{100}$ gr. of hyoscin and $\frac{1}{6}$ gr. of morphin, after which under alypin anesthesia I removed the ethmoidal cells and opened the frontal and antrum into the nose, removing the dead bone which extended from the alveolar process into the antral floor.

The laryngeal patient was talkative and remembered some of the operative incidents, but not in an unpleasant way. A sinus patient had complete amnesia.

I have used this method in a little over one hundred cases in private practice. About 75 per cent. of the cases have been for submucous resection, and the rest of the cases for various conditions of the ear, throat and larynx. I have not used this method for any tonsil operations.

The dosage has ranged from $\frac{1}{100}$ gr. of hyoscin and $\frac{1}{8}$ gr. of morphin to $\frac{3}{100}$ gr. of hyoscin and $\frac{1}{24}$ gr. of morphin. In

about 60 per cent of the cases $\frac{1}{100}$ gr. of hyoscin and $\frac{1}{8}$ gr. of morphin sufficed.

I appreciate that this is a limited number of cases from which to draw definite conclusions, and as the recorded results of others are not of a similar class of cases or of like dosage, it is hard to draw any conclusion. Most of the fatal cases show grave visceral lesions, extremes of age or enormous dosage.

I do not advocate this method as a routine for all cases, as we are using two powerful drugs. The phlegmatic patient will do well under local anesthesia alone, while the neurotic patient who turns pale and is nauseated during a painless examination, will hardly endure an operation with all of their special senses overactive, even if the operative field is free from pain, as there will be a nervous shock which can be prevented by using the above method.

DISCUSSION ON PAPERS OF DRS. KYLE AND DAVIS *

DR. J. J. KYLE, Los Angeles: I am sorry I have not had more experience in the use of hyoscin and morphin. Dr. Hurd reports a series of one hundred cases, and after such a trial as that we know his investigations are worth something. When we see the great enthusiasm of the general surgeon, and more particularly the abdominal surgeons, on the use of morphin and hyoscin and the system of blocking the nerves, I am more than convinced we have not paid as much attention to this form of anesthesia as we should. I think sometimes the question of shock depends on the anesthetist to some extent. Sometimes he goes at the patient with a degree of roughness sufficient to bring about a nervous disturbance. Some seem to show a knowledge of psychology of the giving of an anesthetic, while others show no knowledge at all. I have an idea this great war will give us some knowledge of the influence of fear and exhaustion on the repair of wounds, and when that literature comes to us we will be able to form a better idea, probably, of the value of morphin and hyoscin.

My experience with morphin and hyoscin has been limited to laryngeal cases and external operations for removal of the larynx, and in these cases I was perfectly satisfied with the results. In malignant cases, and in women who are frightened, I can understand it would be better if we could take the patient to the operating table and operate with him unconscious of the act. I think the method would be indicated in operating on tubercular mastoids, particularly where we have advanced pulmonary lesions. Most of these patients die, however, after operation.

DR. J. W. MURPHY, Cincinnati: I shall speak of Dr. Davis' paper first. I am satisfied all who have used cocain in any strength recognize its toxicity. Certain patients will show bad effects from its use in any solution, be it saturated or as low as $\frac{1}{2}$ per cent. Perhaps no drug has received so much attention and experimentation as cocain, the different strengths that will give anesthesia and the different technics in its use. I have been experimenting, and I think with all solutions we have I have seen some unfavorable symptoms, but I have settled down now to the method as advocated by the essayist, and use altogether a saturated

* The paper of Dr. Davis will be found on page 302, Oto-Laryngological Section.

solution. I have been using cocain crystals for ten years, carefully applied, having moistened the cotton with adrenalin, only having sufficient on the applicator to produce the effect without any excess running into the throat and being swallowed. I believe the saturated solution gives less toxic effect than any other solution. I have used it so frequently that it has become a routine practice. I use it the same way in nasal work. I have not followed the essayist's method of applying it on the posterior turbinate body and allowing it to remain for some time. I think our earlier method, where we had so much trouble, allowing the patient to sit in the anteroom for an hour and a half, is where we got our bad effects. Now I do not allow the patient to wait. The anesthetic is so much more rapid with the saturated solution, I am satisfied we get better results than with the weaker solutions. I find the local application to the nostrils of cocain is insufficient and have not attempted to cocainize the tonsils through the nose. I apply cocain to the mucous membrane and use a solution of hyoscin as advocated by the former speaker. In tonsil operations I do not depend entirely upon cocain but use novocain. I find just as good results in $\frac{1}{2}$ per cent. as Dr. Kyle claims for 2 per cent. It seems to give just as good results.

With reference to Dr. Hurd's interesting paper, I do not know of anything that has given me more comfort in operative work than the use of hyoscin and morphin. I believe that is the same as scopolamin. That is also a recent practice, even for nasal and throat work. If you can remove the first element of fear in your patient you have gone far toward success in your operation. With careful application of this you can do so. Certain cases have an idiosyncrasy against scopolamin and you fail of results. I have felt that this came from the drug being in a tablet form, and I have had a solution put up so that 10 drops represent $\frac{1}{150}$ of scopolamin and $\frac{1}{8}$ morphin. This can be varied to give any dosage you wish. This is better than a tablet where there is an element of error. I never use it in children and it should be given one hour and a half before operating.

DR. JOS. C. BECK, Chicago: I feel really indebted to the gentlemen for presenting these papers and also to the program committee for putting such a subject before us for discussion. I know that what I am going to say will not appeal to many here, but I have practically abandoned local anesthesia wherever I can. In any operation that requires an anesthetic at all I use the morphin and at times hyoscin as preliminary and follow the teachings of Crile in putting the patient to sleep by general anesthesia. I do not mean a deep sleep, but the use of gas or ether with the open-mouth method. It is remarkable what results we have in avoiding immediate shock, and the postoperative neurosis, as the internists call it. I think this will go a great way in having our patients better satisfied than by the local method of anesthesia.

With reference to the laryngeal operation with scopolamin, he said the patient spoke. I would like that explained.

It behooves all men who do work about the throat and mouth to recognize the work of Gwathmey on the use of rectal anesthesia by means of ether oil. It works beautifully. Six ounces of ether and two ounces of oil is approximately the amount used in an adult.

DR. J. D. HEITGER, Bedford, Ind.: A number of years ago in one of the clinics we used hyoscin considerably and occasionally found that it produced just the opposite effect of what we desired in that it excited the patient. By combining a small dose of apomorphin, about $\frac{1}{40}$ grain, just enough to relax the patient, we were often able to get the desired result and thus overcome this embarrassing factor.

DR. LINN EMERSON, Orange, N. J.: As Dr. Hurd has said, one of the objections is the difference in the drug made by different manufacturers, and the marked idiosyncrasy of different individuals. It comes home to one who has had the unfortunate experience I have had. I had an old gentleman on whom I did a cataract operation a few years ago. He became noisy at night, and the intern gave him a hundredth grain of hyoscin and repeated in two hours; the old gentleman promptly died. Oculists who give scopolamin find that unless they are very careful the patients get a marked physiological effect therefrom. For that reason I have felt safer in tonsil work in giving ten minutes before operation begins a full dose of morphin hypodermically. It quiets the patient very much. If you get an overdose you do not feel the alarm and concern that you would if they showed untoward effects from hyoscin. The plan suggested by Dr. Hurd requires the giving of so much time, and many of us are not so situated that we can be called for from three to five hours.

DR. JOHN LESHURE, New York (read by Dr. Hayden): The subject presented by Dr. Davis is one of prime importance, for the physiologic action of drugs so generally used as cocain and adrenalin should be better understood.

In the article referred to by Dr. Davis I incorporated the results of some animal experimentation which was undertaken to determine the effect of strong cocain-adrenalin solutions on living blood-vessels.

The subjects for demonstration were tadpoles about 30 to 35 mm. long.

When of this size these animals have a thin, membranous, lateral outgrowth from the caudal appendage. This is highly vascular, and each half is supplied by branches from the aorta and central vein of the corresponding side, which pass down the thick central stem. The point of practical importance is that there is no direct communication between the blood-vessels of the two sides.

It is possible, therefore, to compare the results obtained by simultaneously applying drug solutions of different strength to corresponding portions of the structure referred to, which resembles in many respects a mucous membrane.

After being curarized, the animal is placed upon a microscope stage and one side is treated with strong cocain-adrenalin solution, the opposite side with 4 per cent. solution of cocain in adrenalin.

Comparison of the two sides shows that slowing of the blood stream and venous stasis occur at a much earlier period on the side treated with the first-named solution than on that treated with the weaker solution. In about twenty seconds the circulation in the smaller vessels has practically ceased. The tadpole, being a gill breather, at this stage of its existence cannot be kept alive more than five or six minutes out of water, but control tests made with uncocainized animals showed that death occurred as early in these individuals as when cocain was used.

This fact would seem to prove that general absorption could hardly have taken place, since cocain is a powerful cardiac paralyzant and would have caused death promptly had it entered the general circulation.

These demonstrations, I think, prove the statement made by Dr. Davis that "the more concentrated the cocain-adrenalin solution the more quickly and completely the local circulation is blocked, absorption into the general circulation is prevented and toxic effect of the drug is obviated."

There is one point which Dr. Davis did not mention, but which I believe he did refer to in his previous article, viz., the importance of giving the patient morphin and hyoscin before cocainization.

Crile in his anoci-association methods uses these drugs, and I believe the dangers of shock and hemorrhage are greatly diminished by their use.

I have not yet had the opportunity of testing Dr. Davis' method for anesthetizing the tonsillar region through the descending branches of Meckel's ganglion, but I believe it should be a most valuable addition to our ways and means and one free from danger if used with the precautions suggested by him.

DR. EDWARD L. MEIERHOF, New York: I wish to add my experience to that of Dr. Davis' in the use of concentrated solutions of cocain. I first saw Dr. Freer of Chicago use cocain in this strength: Three to four grains of anhydrous flake cocain hydrochlorid are dissolved on a cotton-wrapped end of an applicator previously soaked with a solution of adrenalin 1:1,000. Rubbing this mop over the septum on both sides for septum operations of any kind will give complete anesthesia, or over any area in the nasal cavity will enable one to do any operation with the minimum of sensation, such as no method that I have tried or witnessed in the work of many others. I have had some patients feel faint, but I have seen no signs of cocain poisoning, such as dilated pupils, sweating or weakened pulse. I do not, however, refuse to give a small dose of whisky or some black coffee occasionally. I have had no severe reaction follow the use of cocain in this manner. I have tried it in tonsillectomy, but owing to traction upon the tonsils it was not so satisfactory. I would not advise its use in eye-work, as it causes marked drying of the cornea.

DR. CHARLES LUKENS, Toledo: When I first stumbled onto a preliminary hypodermic of morphin and hyoscin as an adjuvant to local anesthesia five years ago, I was delighted. It was one of the best things I had discovered and continued use of this procedure has been most gratifying, so that it has become a routine with me in all cases of nervous people on whom I expect to do any particular or trying work under local anesthesia. I have been peculiarly pleased with the effect on the unruly cataract patient, and in glaucoma, where ether would ordinarily be required, and my advancement cases are made much more comfortable by its use. I have had two unpleasant, but not dangerous, effects where I had to postpone the operation, one vomiting (cataract man) and one collapse of a young woman. My usual orders are that $\frac{1}{8}$ to $\frac{1}{4}$ grain of morphin with hyoscin be administered forty-five minutes before time to start local anesthesia.

DR. HOWARD F. PYFER, Norristown, Pa.: In a small town if the surgeon gives pain to his patient during an operation, the fact becomes the knowledge of future victims, producing fear and anxiety and in many cases deferring necessary operative work. Recognizing this, I tried hyoscin and morphin, then scopolamin and morphin. The first, clinically, I believe to be the better, though pharmacologists think they are alike. Though, as a rule, this injection was efficacious, unfortunately it produced nervous hysterical attacks that were annoying, especially if seen by anxious relatives. So I abandoned these preparations and now use morphin and atropin with no bad effects, no shock, and no pain. However, such an injection should not be given preliminary to the administration of ether. In local anesthesia granulated cocain is easily poured from the bottle on the adrenalin solution, making the Freer mud. If this preparation is used on the tonsils and adenoids, perfect anesthesia

results, but the disagreeable hawking and spitting are annoying and distressing to both operator and patient. Injection of adrenalin in tonsil work is objectionable, for secondary hemorrhages following its use are not rare.

DR. OLIVER TYDINGS, Chicago: Starting in the practice of medicine before the days of cocain, I have used it since 1887 and I saw the effects of cocain poisoning before I ever saw cocain. I will say that the element of fear is the most dreaded thing that concerns our patient. Before the introduction of this, in general anesthesia days, I learned to use morphin and atropin, and I learned of that personal equation we have to contend with in the use of hyoscin. Sometimes you get a delirium that is not pleasant for the friends to look upon. I use cocain as the safest and best of all local anesthetics, and if I have ever seen a case of cocain poisoning I did not know it. My regular formula in the removal of tonsils is $1/10$ of a grain. Years ago I said $1/3$. One-tenth divided between the two tonsils, 10 to 20 minims of adrenalin chlorid, water \mathfrak{z} ij. I have seen in my tonsil work but one case of hemorrhage following removal of tonsils since 1904, and I have not removed a tonsil without using adrenalin. In that one case hemorrhage followed immediately after operation. If you use the weaker solution you are using it strong enough. You can produce all the anesthesia you want and will not be afraid. I use cocain mud as taught by Dr. Freer. When I want to operate on a limited field, I use this method, and with it you will not get cocain poisoning.

I always have my patients in the reclining position while operating on the nose.

DR. G. W. MCKENZIE, Philadelphia: There is one simple procedure to prevent shock and at the same time quiet the patient. Have the patient take a good big meal before a local anesthetic is used. Soldiers fight better on a full stomach. Tobacco smoke is a quieting thing for a person accustomed to its use, also a cup of coffee. Do not forget amyl nitrate.

DR. HURD (closing discussion): Replying to Dr. Beck's question, from the time I saw this patient he talked and from the time I split his cartilage he talked all the time. I could not understand him, of course, and he seemed surprised I did not answer him.

Dr. McKenzie asked about the exciting stage of morphin. I give it about forty minutes to an hour before operation, and this is over before the operation. I found after thirty to forty minutes the patient became drowsy. Much is said about cocain as a poison. I think we get our patients ready for operation and they go into a syncope from fright and fear. They have acute shock, due to fear. I would suggest to the men using cocain to try alypin, and you will not get the stage of mental exhilaration and you will not get the after-effect of cocain which is a depression that may last twenty-four hours. It is just as anesthetic as cocain.

OBSERVATIONS ON THE TOPICAL DIAGNOSTIC
AND PSYCHIATRICAL VALUE OF THE
WILBRAND TEST WITH A NEW
CLINICAL INSTRUMENT *

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For ten years following the original suggestion of the topical diagnostic possibilities of the hemianopic prism phenomena by Wilbrand in 1899, little interest was taken in the question, as is evidenced by the lack of communications on the subject until that of Behr in 1909. Apparently it was the investigations of Hess, in 1907, on the allied subject of the hemiopic pupillary reaction, which stimulated interest, for the two subjects have since that time usually been considered together.†

Wilbrand,¹² in 1899, having become convinced that the Wernicke hemiopic pupillary reaction was a practical failure, decided that a new method to the same end was desirable, and proposed his hemianopic prism phenomenon. With one eye covered, the hemianopic patient was told to concentrate attention on a small white point on a black wall. A strong prism was then suddenly placed before the eye, throwing the image on the blind retina. If the eye instantly moved so as to fix on the point again, the lesion was central; otherwise it was peripheral to the basal stem ganglion. Likewise, if the prism was suddenly interposed, throwing the image on the seeing retina, the eye instantly moved to fix the image again; but when the prism was suddenly removed the eye would once more jerk back to fix the point or not according as the lesion was central or peripheral. While he found positive or central cases verified at necropsy, he found no negative or peripheral cases. One case of acromegalic bitemporal hemianopsia failed to give the negative test, presumably, he thought, because there was some light perception left in the defective field. The test may be made binocularly in homonymous cases. Wilbrand himself did not

* From the surgical clinic of Prof. Harvey Cushing at the Peter Bent Brigham Hospital, Harvard Medical School.

† Thus both subjects were considered in our communication⁹ of 1913. But this year the data and instruments have become too bulky for a single communication. The instrument here presented could not be satisfactorily transported and was reserved for this session. The new instrument for testing the hemiopic pupillary reaction has already been presented elsewhere.¹⁰

argue the presence of a definite reflex arc in this phenomenon, but subsequent observers have postulated such an arc; for instance, Behr locates the afferent branch in Bernheimer's fibers, which are supposed to have about the same course as the pupilomotoric fibers.

In the latter part of 1906, Bielschowsky⁴ reported a single case, that of an intelligent young girl with temporal hemianopsia in one eye and blindness in the other eye from a chiasmal lesion. Refixation was equally prompt on each side with 40-degree prisms.

Behr,¹ in 1909, reported the Wilbrand test on twenty cases of hemianopsia, by the prism method, and found no reason to doubt the diagnostic value of the test. In the same year, Lenz,⁸ in an exhaustive study of the cerebral visual paths, reviewed the work of Behr, but ventured no opinion in the absence of sufficient pathological material.

In 1910 Krusius⁷ examined four cases by the prism method and reached the conclusion that the test was of doubtful value. A little later in the same year Köllner⁶ reported the Wilbrand test in nine cases of homonymous hemianopsia, all presumably of central origin. Binocular prisms of 15- to 30-degrees were used. The considerable delay shown by these patients in refixing the object on the blind side led him to doubt the diagnostic value of the reaction. Jess,⁵ in 1912, arrived at the same conclusion from a study of three cases.

In my own study¹⁰ of twelve cases, reported in 1913, I came to the conclusion that several important factors had not previously been sufficiently well controlled. When prisms are used, unless they are placed before the eye very quickly and carefully, the patient may note a slight movement of the test object, indicating in which direction the object has apparently been deflected. The position of the thick or thin edge of the prism may be associated by an intelligent patient with a certain position of the test object. But worse than this, the test object always returned to the center when the prism was removed, and had always been placed so as to refract in the horizontal plane, so that intelligent observation on the part of the patient could materially aid him on repeated trials. And queerly enough the crucial experiment had never been recorded of using a disappearing object and noting the action of the eyes when the object was simply lost from view and no stimulus was active in the blind field. This latter test threw considerable light on the

intelligence of the patient as a complicating factor. To control all of these possible complications, from one to three small electrically lighted spots were used. These were invisible to the patient and could be moved into various planes and distances from the center. All could be turned on or off at will in any sequence. Although this method required the use of an assistant to move the lights into different positions—for the patient was to be entirely denied advance information as to where the lights would next appear—nevertheless very consistent results were obtained, and all for the most part cast great doubt on the diagnostic value of the Wilbrand test. However, it seemed possible that if the reaction times were carefully studied in a large number of cases with an elaborate apparatus meeting all the requirements, certain characteristics of the reaction could perhaps be found which would be of practical, or at least scientific, value. The apparatus previously used was somewhat difficult and time-consuming to manipulate and could be improved in certain features. Accordingly a new instrument was designed and built, with which a large number of patients could be easily and quickly examined.

THE NEW INSTRUMENT

In order to obtain displacements of the test lights up to 45 degrees from the center and not vary this distance from the patient, a large awning or canopy umbrella, five feet in diameter when extended and built on a radius of one meter, was selected. This was painted black and mounted on an adjustable stand. In order to present a smooth concave inner surface to the patient the center pole and bracing were removed and the umbrella was held open in a vertical position, as shown in the cut, Figure 1, by guy wires running from each rib behind to a corresponding steel spoke extending from a forged central hub attached to the top of the adjustable stand. These spokes in addition support a board 12 inches wide by 14 inches long, which forms a central station for the wiring system. To this board are also attached a tin box containing three dry cells, a resistance coil, and an automatic magnet short switch. The latter is packed in a heavy felt covering to muffle any slight click that it may make.

From the central station a piece of rubber gas tubing 10 feet long, containing twenty-four wires for the necessary connections, runs to the portable switchboard, which has a simple push button key for each light and a resistance coil or rheostat

for regulating the intensity of the lights. The central switch on the switchboard for controlling the central light on the umbrella is arranged differently than are the switches for the other lights. When it is released it springs upward and meets a contact point, lighting the central light directly until any other switch is closed, when its corresponding lamp will light



Fig. 1.—Photograph showing general arrangement of patient in position to be examined with the new instrument. Apparatus is slightly turned to give better view.

and the central light in the same instant be automatically turned out. If the central switch is pushed down away from the upper contact the central light goes out. If now it is pushed still further down, until it strikes the lower contact point, the central lamp is again lighted in the same way that the other switches light their respective lamps.

ance (r) and the armature of the magnets (M) to the battery (B), as shown in the cut. The resistance (r) is equal to that of the magnets (M), so that the light intensity is the same in either circuit. The contact (C) may be rotated to one side if its use is not required. The other contact (No. 10), below, lights the central lamp through the magnets as do the other switches.

The operation of the lights by this arrangement is obviously simple. In the position shown in the cut, the central lamp only will be lighted, the current passing from the battery through the armature of the magnets and the resistance (r) through the contact (C) and the central switch to the central light and back to the battery through the rheostat (R). If now any other switch is closed the current shunts through the magnet (M), which draws down the armature, breaking the circuit of the central light, and at the same instant the whole current passes through the magnet around through the wires and lamp corresponding to the switch closed. If the switch is released the central lamp will again light, as the magnet allows its armature to spring up to its contact point. If it is desired to turn out the central light momentarily, the central switch is simply depressed until contact with (C) is broken. Further pressure will relight the central lamp as the switch touches contact point No. 10. If any other lamp is burning and it is desired to turn it out without lighting the central lamp, either the contact (C) may be rotated to one side or the central switch may be pressed down, out of contact, and the switch corresponding to the lighted lamp released. But if we do not wish the center to be relighted each time but wish to change the light to various parts of the field, the contact (C) is turned to one side and each switch lighting a lamp is released as the next one is depressed. In this way displacements up to 90 degrees may readily be obtained.

The lights controlled in this manner are arranged on the umbrella in the horizontal line and in the two lines at 45 degrees with the horizontal passing through the center. There are three lights distributed along each line from the center, so that displacements of 15, 30, and 45 degrees may be obtained in each direction from the center when the patient is seated one meter from center. The lamps ($2\frac{1}{2}$ volt, tungsten) are set in a large cork, as shown in (L) of Figure 2. The cork was first cut perpendicular to its axis through the central point. Each half

was then bored from each end with different-sized cork borers until a cross-section, such as is seen in the cut, was obtained. The lamp, with wire connections soldered on, was then screwed into the upper segment and a disk of milk glass fitted into the lower segment, as indicated in the drawing. Each lower segment was then glued in place on the back of the umbrella and a hole burned through the cloth with a red-hot iron rod corresponding to the hole in the cork, at the same time blackening the inside of the cork. The milk glass disk was then placed in position and the upper segment containing the lamp was glued in place over the lower segment. The purpose of having the light surface thus set at the bottom of a black tube about 1 cm. in depth is to render it entirely invisible to the patient in semi-darkness, except when illuminated.

For convenience in manipulating the lights, the switchboard was made about the same size as a field chart and the keys were arranged in the same relative position and order as the lights on the umbrella. In this way the keyboard may be operated with one hand entirely by touch, and undivided attention may be given to observing and timing the movements of the patient's eyes.

METHOD OF EXAMINATION

Since we are studying a reaction which is supposed to have characteristics of a more or less involuntary or subconscious nature, it is obviously necessary to eliminate as far as possible thoughtful and voluntary processes. To get the particular mental repose which would be ideal for these examinations is very difficult, if not impossible. It is not desirable to instruct the patient as to the method and rationale of the examination, and yet this very ignorance of the procedure has often been noted to make the patient fidgety and querulous in regard to what is taking place, at a great expense to their mental repose. Difficulties of the following sort arise. The patient has had several fields taken and possibly other examinations requiring rigid central fixation, no matter what takes place in the peripheral field. Now we do not wish the patient to stare wilfully in one direction in this examination, nor do we wish to suggest that the field be deliberately searched for the test object in case it should disappear. The examination should not be attempted on the same day that a field or other test requiring central fixation has been made, and preferably should be made in another room, to dissociate as much as possible from the

idea of perimetry. But even at best the patient, if intelligent, rapidly becomes familiar with the procedure as it is repeated, markedly reducing the refixation time, and finally, as facility increases, may become interested and amused at the "hide-and-seek" element of the examination. Thus it becomes necessary to conduct the examination in two stages. In the first stage the patient is given as little instruction as possible and is quietly seated before the instrument, without unnecessary distractions of any kind. He is simply told to observe the light wherever it may be. The automatic return to the central light is used only at intervals, since there is a marked tendency on the part of the patient to look back to the center if it is used continuously. At the end of the first stage (five to ten trials) he usually begins to show a greater or less decrease in reaction time, or if he is not very intelligent further instructions are necessary to gain a better state of mental repose.

In performing the tests the patient is seated at a distance of one meter from the center of the apparatus. While a chin-rest is not necessary, it reduces the tendency to turn the head with the eyes, thereby making observations of ocular movements easier. There should be dim room illumination, just sufficient to show clearly the movement of the patient's eyes. This illumination is from a rheostat ceiling light just out of the patient's peripheral field of vision above. Observation of ocular movements may be made with a short-focus telescope or by the unaided eye. No difficulties are presented either way. The intensity of the test lights is regulated by the rheostat to the point where no sense of dispersion light is obtained when its image is made to fall on the normal blind spot. Such an intensity of illumination, while greater than that of a white spot on a black wall, still offers no indication by dispersion sheen to the hemianopic patient of its location when it is in the blind field. In case there is a relative central scotoma the examination can usually still be made if there is vision in the neighborhood of 20/200, by increasing the light intensity somewhat, since with macular damage the sensitiveness to dispersion light is not so acute.

For rapid and accurate manipulation of the lights with respect to the field of vision a copy of the field may be held close at hand, or the field outline may be lightly chalked on the black top switchboard for each eye, so that one may simply press a key in a certain part of this charted field and a light

will appear in the corresponding field of vision of the patient. When the reaction runs over a second it should be timed with a stop-watch. If the reaction time is less than a second, a system of counting, carefully practiced, is more satisfactory.

In taking the end point of the reaction time it has been found much better to watch the eye come to rest rather than to ask the patient to say some such word as "Now" when the light is located, because the patient seems to feel more under strain of examination with this added requirement and is apt to put much more wilful effort into locating the spot, immediately showing an exaggeration of the tendency to seeking movements.

Prisms have not been used, for several reasons: (1) The patient may get an inkling as to which way the image is going to be refracted as it travels the edge of the prism, or (2) by associating the thick edge of the prism with a certain position of the image. (3) He may see certain portions of the room through the prism which form orientation marks as to the position of the light. (4) If the prism is held close to the eye, the refraction prevents accurate observation of the eye movements. (5) If the prism is held forward far enough to observe the eye directly, the patient may get either orienting or disorienting glimpses of the room around the prism margin.

Previous experience with these tests having shown that the reaction time decreased after the first few trials, or as soon as the patient became familiar with the general procedure, it became necessary to proceed with the simpler tests first and with these confined to the blind side, for if the light is first changed from the center to the seeing side and then disappears in the center to appear on the blind side the natural conclusion is that it must be in that side, since it is not in the seeing field. Therefore the 15, and then the 30 and then the 45 degrees displacement horizontally is tried on the blind side. The center light is now simply turned out and no light appears on the blind side. Sometimes the latter test may be made first. From this point on the order is not of importance, and a great variety of trials may be carried out in any order.

The method of recording results must be as simple and rapid as possible. Probably the best way is to record the lights by numbers. The patient's chart is outlined on the keyboard. Each key is given a number, which is easily remembered. The displacement is then recorded by the first two numbers and the time by the third number, followed by remarks on the character

of the reaction. Record is kept of the reaction time on the blind side only, since the reaction time on the seeing side is very constant and very short, usually in the neighborhood of one-fifth of a second or less. Before proceeding with the examination the field chart is studied and a large series of numbers, indicating desirable displacements, are put down in columns, so that on observation merely the reaction time and occasional remarks need be recorded.

In all, twenty-six cases have been examined for the Wilbrand reaction by the electric-light method. The space and figures necessary for the report of all these cases would hardly be justified by the results. Therefore, instead of reporting several cases of similar field type and reaction only one typical case is given. The fields were all taken with the instruments and in the manner previously described in another paper.* Only the field for the normal disk is given, since outside of this periphery the test lights are not visible unless close enough to the line of cleavage to be detected by dispersion light.

In the group of anterior lesions the cases naturally fall into three subgroups, according to the character of their field defects: First, those with complete, incomplete, or more than complete bitemporal hemianopsia; second, those with no vision in one eye and complete or incomplete or more than complete temporal hemianopsia in the other; third, homonymous hemianopsia of tract origin. In the group of posterior lesions, subdivision may be made into complete and incomplete homonymous hemianopsia and cases with loss of vision in one eye and nasal hemianopsia in the other eye.

Certain definite types of refixation movements of the eye have been noted in most of these cases. These have been given terms such as pseudorefixation, central refixation, pseudocentral refixation, angular refixation, and pseudo-angular refixation, which will be described in the somewhat more detailed summary of results given with the first case so that thereafter unnecessary and tedious descriptive accounts may be avoided.

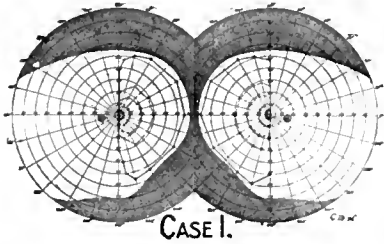
CASE REPORTS

CASE 1. (Surgical No. 435).—Hypophysial struma. September 27, 1912. Mr. H. C. N., aged 42. Failing vision for two years, associated with frequent headaches, diplopia, nausea, and vomiting. Complete bitemporal hemianopsia for over one year.

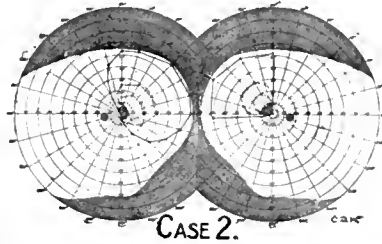
* Walker, C. B.: Some New Instruments for Measuring Visual-Field Defects, *Arch. Ophth.*, 1912, xlii, No. 6.

Examination on admission, typical hypophysial struma with evidence of neighborhood disturbances. Well-educated professional man. Mental lethargy apparently slight.

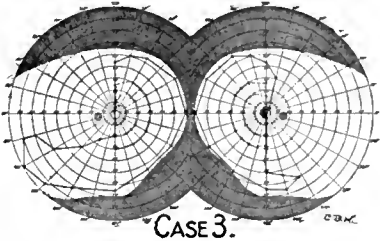
Eye Examination.—No nystagmus or exophthalmos. Transitory weakness of third nerve with pupillary reaction to direct light on right. Consensual reaction from left eye good. Left



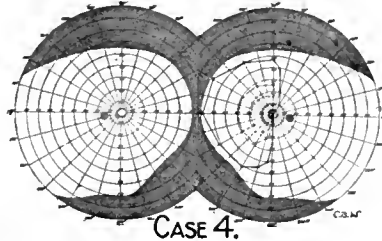
Chiasmal lesion; V. O. D. 20/70; V. O. S. 20/70+.



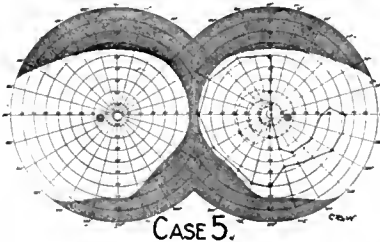
Chiasmal lesion; V. O. D. 16/200; V. O. S. 20/100.



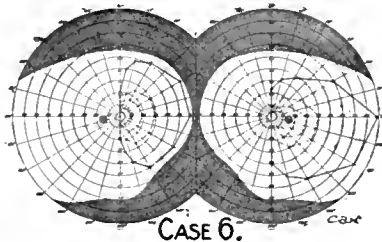
Chiasmal lesion; V. O. D. 20/200; V. O. S. 20/30.



Pituitary tumor; V. O. D. 20/40; V. O. S. nil.



Chiasmal lesion; V. O. D. 20/30; V. O. S. nil.



Tract lesion; V. O. D. 20/40; V. O. S. 20/30.

Fig. 3

Fig. 3.—Typical fields of anterior cases examined.

pupil reacts well to direct light. Hemiopic pupillary reaction marked by rotary shutter test. Right pupil 3.5 mm. diameter, left 4.5 mm. diameter in ordinary light. Field and vision, Figure 3, Case 1.

Wilbrand Test.—O. D.: When pseudorefixation was attempted on the first trial, the patient continued to stare at the center for six seconds, then gave a quick glance toward the

blind side, and then announced that the light was out. On the next trial the 15-degree light was flashed as the central light was turned out. The patient continued to look at the center for four seconds and then gave a quick glance toward the blind side as before, but discovered the light this time and refixed on it immediately. Trying now the 30-degree displacement, the patient immediately looked to the previous displacement and then with a process of seeking found the light. Reaction time two seconds. When the 45-degree light was tried it was found by the same process in three seconds, and on repetition in two seconds. Patient was apparently familiar with the reaction. A light was displaced to 30 degrees on the seeing side. Instant refixation, but when it was turned out and the 15-degree light on the blind side was lighted, the patient's eye instantly sought the center and searched for three seconds before the light was located on the blind side. This we have called pseudocentral refixation. The same thing occurred when no light at all was flashed on the blind side. Pseudorefixation in the horizontal was now marked but rapidly performed, the patient being confident that no light was present in from two to three seconds. Displacements in the 45-degree axis above or below the mid-line gave what we have called angular refixation, that is, the eye moved out along the horizontal axis to the blind side until the light was discovered above or below, when it was instantly refixed. The reaction times did not measurably exceed those for the horizontal line, but became reduced on repetition to one second or less for the 15-degree displacement, to two seconds for the 45-degree displacement. At first there was a marked tendency on the part of the patient to look at the point where the light had last appeared on one axis and then up or down to the point where it actually was on the other axis. There was no tendency to look directly at the light until several trials, when the patient began to show considerable anticipation of the probable point of next appearance of the light instead of looking at the previous point. Pseudo-angular refixation was now present to a marked degree. That is, when the fixation light was turned off, the eye moved to fix a point on one axis and thence to another point on another axis.

O. S.: The first few tests on this eye were interesting, in that the results were obviously complicated by the experience obtained from examination on the other eye. Thus, when pseudorefixation was first tried the patient immediately began to search over his seeing field—that is, this eye moved to the same side as before when the light disappeared. The patient evidently then realized his mistake and searched his blind field. This, however, occupied only five seconds before he announced that there was no light. Subsequent waverings toward the seeing side were noted, but the reaction time in the course of two or three tests were the same as in the other eye in the second stage, with all the phenomena present previously described.

Four other cases (Surgical Nos. 28986 and 29117, J. H. H., and 2 P. C.) with this stage of field defect have been examined, with practically the same result. Those with better central vision refix slightly more rapidly and those with more sluggish cerebation react more slowly. All showed the same type of refixation and pseudorefixation.

CASE 2 (Surgical No. 247).—Pituitary tumor. July 15, 1913. Mr. M. B., aged 43. Vision began to fail three years ago. Diplopia at same time lasting four months. Six months ago dizziness lasting two months. For past four years marked drowsiness during the day. Examination on admission shows a rather typical case of hypopituitarism. Mental apathy well marked.

Eye Examination.—No exophthalmos, nystagmus or muscular imbalance notable. Fundi show very pale optic disks with slightly blurred margins. Optic cup filled in and lamina cribrosa obscured apparently by a layer of new tissue which extends somewhat over vascular trunks. Macular region and peripheral fundus clear. Pupils moderately dilated, 6 mm. diameter right, 5.5 mm. diameter left. Good reaction directly and consensually. Both eyes showed a well-marked hemiopic pupillary reaction on direct examination with the rotary shutter. Reaction to accommodation sluggish. Fields and vision Figure 3, Case 2.

Wilbrand Test.—O. S. In the first test for pseudorefixation the patient began to search about the center after seven seconds, and in three seconds more decided that the light was out. The 15-degree light was refixed on the blind side during the course of a seeking process lasting five seconds. The 30-degree light likewise was refixed in seven seconds, and the 45-degree light in ten seconds. Pseudorefixation now marked. Passing into the second stage by repetition, the reaction was reduced to from three to five seconds for the horizontal lights. Angular refixation almost equally good on upper axis, with marked pseudo-angular refixation present. Refixation time in lower half of field much delayed and variable—ten to thirteen seconds at first—and in the lower nasal quadrant the 45-degree light could be hidden for twenty seconds at first, while the whole upper and lower temporal field was being searched. The various forms of pseudo-angular and pseudocentral refixation were very marked. Toward the end of the examination reaction times had decreased about one-half, and a tendency to systematic search of the blind field was noted.

O. D. Changing quickly to this eye and giving the pseudorefixation test, the patient began immediately to search on the same side as before and announced that the light was out before the blind side had been searched. This tendency to look to the seeing side delayed the reaction times in the first stage so that they were practically the same as in the eye first examined.

Improvement took place in the second stage, but the field being smaller and the scotoma larger, the reaction times were from two to four seconds longer throughout. Another case (S. No. 921) examined at a stage of the disease almost as advanced as in this case gave very similar, though somewhat shorter, reaction times throughout.

CASE 3 (Surgical No. 496).—Acromegaly. Oct. 16, 1913. Mr. C. M. W., aged 24. Very gradual failing of vision for three years. Diplopia first noticed two years ago and has become more persistent from that time. Signs of acromegaly first noted seven years ago. On admission patient found to be a typical case of acromegaly with incomplete bitemporal hemianopsia. Mentally keen.

Eye Examination.—Patient is myopic 1 D., and has used a monacle for one and a half years in the left eye to aid suppression of vision in the right eye and to avoid discomfort of diplopia, due to weakness of internal recti (2 degrees). No exophthalmos or nystagmus. Fundi show pale optic disks, right slightly paler than left, but lamina cribrosa is obscured in a shallow cup. Nasal disk margins hazy, temporal margins sharp. Vascular condition good, macular region and peripheral fundus clear. Pupil in the right eye (4.5 mm.) larger than pupil in the left eye (4 mm.). Good reaction to accommodation and to direct and consensual light. Hemiopic pupillary reaction present with rotary shutter in upper quadrant. Fields and vision Figure 3, Case 3.

Wilbrand Test.—O. S.: On the first test for pseudorefixation it was noted that the patient began to search at once in the upper temporal quadrant for the light, and in two seconds had satisfied himself that the light was out. But it must be noted here that the patient was of an inquisitive disposition and could not be prevented from looking the instrument over on entering the room. Being a mechanic, he surmised at once that lights could be made to appear in different parts of the field. Further, having had suppressed vision in O. D. for so long a time he knew his weakness in the upper temporal quadrant. He took much interest in locating the lights quickly and was highly diverted by the "game" feature. After a few tests the lights were located in a second or less. Of course pseudorefixation, angular and pseudo-angular refixation between the horizontal and the 45-degree axes were present and almost equally rapid. Throughout the examination there was a very marked tendency to turn the head as well as the eyes in refixation, doubtless the result of limitation of ocular motion by the monacle.

O. D.: On the first test for pseudorefixation the patient glanced up into the upper nasal quadrant (homonymous with the upper temporal quadrant of the other eye), and then searched the temporal quadrant, in six seconds announcing that the light was out. Refixation on the lights in the horizontal required from two to four seconds in the first stage and from one to two

seconds in the second stage of the examination. All the previously described forms of refixation and pseudorefixation were readily demonstrable.

Two other cases of incomplete bitemporal hemianopsia, one less advanced and one more advanced than this case, but both having a less active mental condition. The reaction times in the more advanced case were a little shorter than those in Case 1, but of very similar type, because the remaining fields in the lower temporal quadrant gave very little help to the patient. The earlier case had the upper temporal quadrant lost in O. S. and vision of 20/70, and very little damage in the other eye and vision 20/30. There was at first no effort at refixation in the blind quadrant, which gave the patient no trouble and consequently received no attention; only seeking motion about the center was noted. However, as the tests proceeded and the patient began to comprehend the state of affairs, the reaction became practically the same as those in Case 3.

CASE 4 (Surgical No. 789).—Pituitary tumor. Jan. 11, 1914. Miss A. D. S., aged 41. Failing vision began about six months ago, progressing bitemporally to complete blindness in the left eye in a week or ten days. Headache, nausea and vomiting began five months ago, but have been growing less severe since admission. Mental condition only slightly below par.

Eye Examination.—No exophthalmos or nystagmus; movements of eyes free and complete. Fundi show clear but not sharp disk margins. Optic cup moderate depth. Lamina cribrosa sharp. Disks are pale but not glistening. Pallor more marked in the left eye than in the right eye. Vessels, macula and peripheral fundus in good condition. Pupil in the right eye (4.5 mm.) smaller than the pupil in the left eye (6 mm.). Pupillary reaction in the right eye good with pupillary reaction in the left eye nil direct, but good consensually direct light, nil consensually. Hemiopic pupillary reaction present. Fields and vision Figure 3, Case 4.

Wilbrand Test.—Pseudorefixation test. Patient regarded the center steadily for two or three seconds, searched about the center for one second and out on the blind side to about 20 degrees for three seconds, and asked what to do next. On the horizontal refixation at 15 degrees the action took place in the same manner in four seconds, at 30 degrees with seeking motion in five seconds, but at 45 degrees the patient's eye sought the 30-degree region, searched about for a second or two, wandered back to the center, and then out beyond 30 degrees far enough to get the light. Reaction time ten seconds. On repetition the light was found in three seconds. Pseudorefixation in about the same length of time. In the second stage of the examination refixation and angular refixation became rapid, one to three seconds for any displacement, with the various corresponding pseudorefixation phenomena present as usual.

Two other cases of this type showed practically the same findings, except the delay (six seconds) in finding the 45-degree light in the first stage was not so marked in one of the cases (Surgical No. 24). In the other case (Surgical No. 1011) there was a very small patch of weak vision left remaining in the temporal field between 45 and 55 degrees on the horizontal line. There was then in this case instant refixation when the 45-degree light on the horizontal line was used. For the 15-degree and 30-degree displacements refixation was prompt, from one to two seconds, but pseudorefixation, angular and pseudo-angular refixation were marked though rapid.

CASE 5 (Surgical No. 578).—Hypophysial struma. Nov. 13, 1913. Mr. M. L. M., aged 36. Vision in the left eye began to fail temporally two and one-half years ago and patient became blind in about one year. A few months later temporal hemianopsia began in the right eye. On admission patient was found to be in the pituitary group with definite signs of hypopituitarism. Professional man of phlegmatic disposition.

Eye Examination.—No ptosis; movements free and complete; both eyes rather prominent; no nystagmus; myopia in the right eye. Fundus of each eye: disks very pale, especially right, which was glistening white. Margins sharp and clear. Optic cup wide and shallow with well-defined lamina cribrosa. Pupil in the right eye (4 mm.) smaller than the pupil in the left eye (5 mm.). Light thrown on the right eye gives a fair direct reaction and a good consensual reaction to the left eye. Hemipopic pupillary reaction present but weak. Direct and consensual reaction from the left eye nil. Fields and vision Figure 3, Case 5.

Wilbrand Test.—The first test for pseudorefixation was practically negative. The patient sat complacently observing the central part of the instrument, thinking, he said, that something had gone wrong with the apparatus, the idea that the light might appear elsewhere evidently not having occurred to him. As soon as this was suggested and the test repeated the upper temporal quadrant was immediately searched. This served as an example of the difficulty of satisfactorily instructing the patient in the first stage. Refixation of light in the upper temporal quadrant was now performed very rapidly, in from one to two seconds. Pseudorefixation, angular and pseudo-angular refixation, for all lights, except of course those in the lower axis and the 45-degree horizontal light. As soon as the patient got an inkling of the method of procedure, refixation in all its forms was rapid, showing the complicating factors of instruments which occur alike in both anterior and posterior cases.

CASE 6 (Surgical No. 663).—Interpeduncular teratoma. Dec. 8, 1913. Miss K. L., aged 11. Always mentally precocious for her age, with tendency to assume the manners of an adult. Suffering with headaches for one and one-half years. Marked symptoms of dyspituitarism and internal hydrocephalus. Although left hemianopsia and left paresis were developing six months

ago, fields could not be taken owing to dull mental condition, and it was not until after a decompression and a silver tube drainage for internal hydrocephalus had been performed that the mental condition cleared so that the fields (Fig. 3, Case 6) were obtained at this, the fourth hospital admission. Mentally the patient seemed almost if not quite as bright as ever.

Eye Examination.—No ptosis, exophthalmos or nystagmus; movements free and complete. Fundus of each eye: typical picture of low grade receded choked disk with very few evidences of secondary changes. Pupils react well to accommodation, direct and consensual light. Left pupil (6 mm. diameter) larger than right (5 mm. diameter). Fields and vision Figure 3, Case 6.

Wilbrand Test.—O. U.: When pseudorefixation was tested on the first trial the patient blinked, looked at the center again, and after searching about centrally for two seconds, again out on the blind side about 30 degrees, then over the seeing side, then up at the observer, saying that the light was gone. A light was refixed at 15 degrees on the blind side in one and a half seconds, at 30 degrees in two seconds, with the usual seeking motion, and likewise at 45 degrees in four seconds. Pseudorefixation now wide and marked on the horizontal axis. In the second stage refixation for any of the horizontal lights required only one to two and a half seconds. Using the various lights on the 45-degree axis, angular refixation always took place without appreciable increase of time. All the other forms of refixation and pseudorefixation were likewise rapidly performed as experience with the procedure increased.

The tumor in this case was exposed at a subsequent operation (supra orbital route), lying in such a position as to involve the right optic tract. This is the only case of a tract lesion examined. The reactions will be seen to be of about the same nature as those found in similar hemianopsias of posterior origin of about equal duration and mentality. The following cases belong in the posterior group.

CASE 7 (Surgical No. 397).—Right parietal lobe tumor. Sept. 15, 1913. Mrs. C. R. C., aged 44. About eight months ago left homonymous hemianopsia developed and escaped detection until complete. Later headaches. Choked disk ($3\frac{1}{2}$ D.), anesthesia and weakness of left side of body, dizziness, Babinski, etc., led to diagnosis of right-occipital-lobe tumor with cerebellar neighborhood signs. Right subtemporal decompression June 10, 1913. On Sept. 21, 1913, a large tumor was successfully removed from the right parietal lobe by Professor Cushing. Mental condition excellent throughout.

Eye Examination.—Both eyes: no exophthalmos; movements free and complete; no nystagmus; media clear. Fundi show typical picture of receded choked disk of low grade after decompression. With no central scotoma and good macular vision, the left pupil (5 mm. diameter) was definitely larger than the right pupil (4 mm.) in ordinary illumination. Good reaction to

direct and consensual light, slight reaction to accommodation. Hemiopic reaction present in both eyes. Fields and vision Figure 4, Case 7.

Wilbrand Test.—O. U.: When pseudorefixation was tested at the first trial, the patient was noted to search about the center with wider and wider swings toward the blind side until at

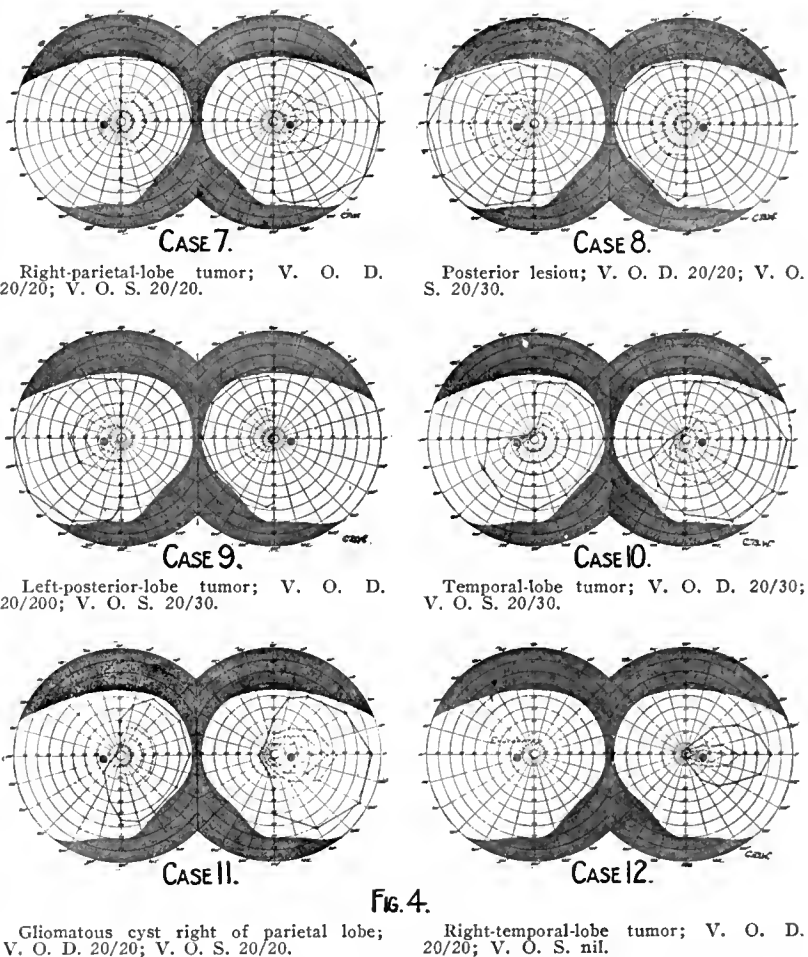


Fig. 4.—Typical fields of posterior cases examined.

seven seconds, with a glance out to about 50 degrees she said she thought the light was not working. The 15-degree displacement was refixed in two seconds, with the same sort of seeking movements as in the first test. The 30-degree light was refixed by a further seeking motion in three seconds. With the 45-degree displacement, however, the patient looked out almost far

enough, then moved the eyes back to the 15-degree and 30-degree region, then up and down, and then announced that the light was out again. On repetition, however, it was found in three seconds, with a more rapid seeking motion, out on the horizontal axis. Pseudorefixation test produced the same movements again, slightly more rapid. Angular and pseudo-angular refixation were rapidly performed now. The light was either found or called out in from one to two and a half seconds in any position. Central and pseudocentral refixation were readily demonstrable.

Three other cases, (Surgical Nos. 29996 and 30418, J. H. H., and Surgical No. 1464, P. B. B. H.) have been examined, with practically the same results, though the reaction times were if anything not quite so short.

CASE 8 (Surgical No. 1441).—June 27, 1914. Mr. G. C. B., aged 37. Severe headaches with nausea and vomiting for past year. Convulsive attacks for five years. Temporary amaurosis for past six months. Occasional diplopia and tinnitus. The neurological examination indicated a left-sided occipital or parietal lesion. Considerable depression of mentality, poor memory, anomia but not true aphasia. Patient understands and readily follows simple directions.

Eye Examination.—No exophthalmos; movements free and complete, but slight nystagmoid movements on looking to extreme right. Pupils practically $5\frac{1}{2}$ mm., possibly right pupil a trifle wider than left. At present no diplopia complained of, and cover test shows no movement. Pupillary reaction active to light, distance, and consensually. Hemiopic pupillary reaction very weak by rotary-shutter test, being just noticeably better when the light is on the seeing than when it is on the blind retina. Fundi show typical high grade of choked disk, $5\frac{1}{2}$ D. in the left, and 5 D. in the right eye. Fields and vision Figure 4, Case 8. Right homonymous hemianopsia; duration unknown.

Wilbrand Test.—Pseudorefixation on the first test was absent, the patient showing no distinct tendency to look to the blind side more than to the seeing side while seeking motions were being performed around a small area about where the light disappeared. A light displaced to 15 degrees on the blind side was not detected until the eye had wandered about the central region for six seconds. As the fixation light reappeared it was practically instantly refixed ($\frac{1}{8}$ second), as was the case throughout the examination. When the 30-degree light was flashed on the blind side it was refixed in five seconds with a similar but more rapid seeking motion. The 45-degree light, however, proved a stumbling block for some time. It was eleven seconds before it was located, most of the time being spent in looking for the 30-degree light. On repetition this light was found in five seconds. The time was not reduced below five seconds for the 45-degree light at any time during the examination, but

the 15-degree and 30-degree lights could be located in one and two seconds as a rule. Angular and pseudo-angular refixation were present but were usually performed in a wavering manner and often required almost a second more time than the corresponding horizontal axis lights. Central and pseudocentral refixations were, however, marked and rapid. This patient never developed the tendency to anticipate the next probable point where the light would appear, but always looked to the point where it had been previously seen. Two other cases (Surgical No. 29395 and 30210, J. H. H.) have been examined, with very similar results, although in each case only one eye could be examined, due to diplopia in one eye and incomplete hemianopsia in one eye of the other case.

CASE 9 (Hospital No. 1046).—Left posterior temporal lobe lesion. Nov. 29, 1913. Mr. L. A. C., aged 22. Headache, weakness and numbness of right arm and leg increasing for two months. On examination patient was found to have a right homonymous hemianopsia, duration unknown; blurred mentality, but directions readily followed; motor aphasia, left paralysis and paresthesia, headaches, choked disk, etc., leading to a diagnosis of a left posterior lesion. At operation three achinococcus cysts about the size of walnuts were removed from the posterior part of the left hemisphere.

Eye Examination.—No exophthalmos, nystagmus, diplopia or muscular imbalance was made out. Fundus of each eye: typical choked disk, 3 D. on the right and 4 D. on the left. Pupil of the right eye (5.5 mm.) larger than the pupil of the left eye (4.5 mm.), with good reaction to light and accommodation. Hemianopic pupillary reaction marked in both eyes with rotary shutter. Fields and vision Figure 4, Case 9.

Wilbrand Test.—Pseudorefixation or actual refixation on the blind side. The patient stared stupidly in the direction where the light had disappeared, although refixation when tried on the seeing side was prompt, about one-fifth second for the 15-degree and 30-degree lights and a scant two-fifths for the first appearance of the 45-degree light. It was hoped that possibly this patient would show some signs of a reflex refixation, but none were forthcoming. It was only after the patient was made to understand that the light might appear on the blind side that there was any tendency to search the blind side. The 15-degree light was then found in eight seconds and the 30-degree light in eleven seconds, but apparently the 45-degree light would not have been located if it had not been suggested that it might be still farther out. Repetition reduced these times to three seconds for the 15-degree light, four seconds for the 30-degree light and six seconds for the 45-degree light. Angular refixation and pseudo-angular refixation required a fraction of a second longer than the corresponding refixation in the horizontal axis. The tendency to look in the direction where the light was last seen was not marked in this case; the eye usually wandered

out in a vague, zig-zag manner and then quickly refixed when the light was noted above or below central and central pseudorefixation was marked. The reactions in this case showed the most marked retardation of any that have been examined.

CASE 10 (Surgical No. 968).—Temporal lobe tumor. Feb. 27, 1914. Mrs. E. A. J., aged 43. Diplopia and occasional headaches for over six months. Slow progressive tendency to left homonymous hemianopsia, beginning in upper quadrants, for six months. On operation, right subtemporal decompression, about 50 c.c. of what was apparently cystic fluid was withdrawn by needle from the second temporal convolution, leaving the temporal lobe collapsed.

Eye Examination.—Slight exophthalmos in both eyes, possibly greater on right. Considerable weakness of right external rectus. Fundus of each eye: increase in venous engorgement and tortuosity, with tendency to partial embedding at disk margins nasally. Margins, especially nasally, are edematous and elevated $1\frac{1}{2}$ D. Picture, if anything, more aggravated on right. Pupils equal (5 mm.), react well to light both directly and consensually. Hemipic pupillary reaction weakly present in quadrant of maximum defect by rotary shutter. Reaction to accommodation present but not marked. Fields and vision Figure 4, Case 10.

Wilbrand Test.—O. S.: As an early case, it was interesting to note that on first test for pseudorefixation there was no tendency to search the blind quadrant on first trial, though the patient looked about the central region for three or four seconds before declaring that the light was out. She had never bothered about the field defect and was concerned only about her central vision and particularly the diplopia. The 15-degree light was located in the blind quadrant in three seconds during the seeking motions about the center. The 30-degree light was apparently deliberately looked for and found in two seconds, and likewise the 45-degree light in three seconds. Pseudorefixation in this quadrant was not marked. By using 60-degree and 75-degree displacements angular refixation and other forms of refixation noted in the cases could not be demonstrated. The examination of O. D. gave the same results as the second stage in O. S.

CASE 11 (Surgical No. 1148).—Gliomatous (?) cyst of right parietal lobe. April 13, 1914. Mr. F. B., aged 30. Severe right-sided headaches, frequently accompanied by nausea and vomiting, for past six or seven weeks. Vision and hearing seem less acute during headaches. At the neurological examination a right-sided lesion was diagnosed. On April 16 a right osteoplastic resection with decompression was carried out and a large gliomatous (?) cyst was evacuated.

Eye Examination.—O. U.: No exophthalmos or lack of parallelism, no ptosis, slight nystagmoid jerks at extreme right and left. Pupils, left (5 mm.) slightly larger than right (4.8

mm.). Both react well to light, accommodation and consensually. Hemiopic pupillary reactions definitely present but not strong by rotary-shutter method. Fundi show typical choked disk of 4 D. elevation. Fields and vision Figure 4, Case 11.

Wilbrand Test.—O. U.: This case, as one of incomplete hemianopsia, represents a stage intermediate between the last two. Pseudorefixation as noted in the first test showed a series of wide oscillations extending to about 40 degrees on toward the blind side. Refixation of the 30-degree light took place in four seconds with the same sort of searching motion. The 45-degree light was refixed in almost four seconds, also probably due to previous test. Pseudorefixation was now marked and rapid. In the second-stage tests all refixations were rapid, from one to two seconds. The various forms of refixation and pseudorefixation were readily demonstrated. As in all other cases there was no tendency to look directly at any of 45-degree axis lamps, except for an evident attempt toward the last to anticipate the next position. Another case comparable to this case in all respects (Surgical No. 30210, J. H. H.) has been examined.

CASE 12 (Surgical No. 1049).—Right temporal lobe tumor. March 18, 1914. Mr. A. D., aged 26. Central vision in the right eye first noted to blur eleven months ago, followed by slow failure of nasal field. In last few months temporary amaurosis has developed and continued with increasing frequency. Headaches, nausea and vomiting frequent but not as aggravating as usual with the fundus a picture of typical high-grade choked disk. Occasional convulsions, uncertain gait, hallucinations and loss of memory.

Eye Examination.—O. D.: Slight exophthalmos; no ptosis or nystagmus; movements free and complete. Typical choked disk with a few punctate hemorrhages and exudates. Macular region peripheral. Fundus clear. Pupil moderately dilated (6.5 mm.) in ordinary light. Reaction to light and accommodation good. The direct pupillary test with the rotary shutter showed perhaps the most striking and characteristic hemiopic pupillary reaction in the series. Fields and vision Figure 4, Case 12. Left eye enucleated thirteen years ago.

Wilbrand Test.—The reactions in this case are interesting as those of an advanced stage probably of homonymous hemianopsia, if the left eye could be properly represented. In the first test for pseudorefixation it was noted that the patient looked immediately to the blind side by a series of jerking horizontal motions, extending out to about 30 degrees. Refixation in the horizontal line was rapid, one second for the 15-degree light, one and a half seconds for the 30-degree light, and three seconds for the 45-degree light. Pseudorefixation was now very rapid, the eye moving out to the 45-degree light in three seconds or less. The tests for angular refixation, however, differed decidedly from those in previous cases. The 15-degree

lights above and below were detected in one second in the usual manner, also the 30-degree light below, but the three other lights were detected in a variable manner. Occasionally they would be quickly detected and angular refixation take place. But this was evidently the result of an accidental peripheral glimpse of the light in the course of seeking motions. For the most part the 30-degree light above and the 45-degree light below escaped detection for from five to ten seconds. The 45-degree light might be found in eight or ten seconds, or the search might be given up. The tendency to look to the point of last appearance was not marked in this case. The motion of refixation was usually a sort of zigzag seeking motion out to the horizontal axis.

SUMMARY

From a study of our series of hemianopic cases with this instrument many interesting though not practical characteristics are notable. Thus it is notable that certain factors may increase the reaction time in any case, and that the presence of a relative central scotoma may add two or three seconds or more to the horizontal reaction time but does not contribute to much delay in the angular reaction time, perhaps because the peripheral retina along the cleavage line sees the light first, after which direct fixation is very rapid. Again, the reaction time in all cases is increasing by introducing variations from simple horizontal displacements, such as angular displacements, change of center, and the introduction of pseudorefixation tests. Further, any patient with a very poor memory or with depressed thinking or reasoning power shows an increased reaction time. On the other hand, in all cases the reaction time for any displacement may be markedly decreased on repetition, no matter in what way it is made. Also, in this group patients with bright mentality and good central vision always gave the shortest reaction time. In all cases there is a decrease of reaction time with the increase of the displacement, but on account of the decrease of reaction time occurring with repetition it makes a difference in what order the displacements are made. Thus if the small displacement is made first, difference in time reaction is not nearly so marked as when the larger displacements are made first. For this reason it seems fairer to time the small displacements first.

Regarding the phenomena of pseudorefixation, certain general statements applicable to all cases may be made. If the pseudorefixation test is the very first test made, the results are quite variable, but at any time later in the trials the results are

very constant. When it is the first test, the patient may simply stare at the point where the light disappeared for from five to ten seconds, give a cursory glance to the blind side, and then announce that the light is out. The more thoughtful group may at once begin to search the blind field over for the light and then give the seeing side a glance before they announce that the light is out. At any time after the first test made on the blind side, the patient almost invariably looks to the point on the blind side where the light was last seen, when the fixation light is simply made to disappear without appearing elsewhere in the field.

Besides the simple forms of pseudorefixation, we can also elicit combinations and complex forms depending on the mental characteristics of the patient. Thus, after refixation has been performed on the blind side in any axis, and on the next trial another axis is used, the eye turns with more or less of a searching motion to the point where the light was previously seen, and then jerks upward or downward to refix the light in its real position as a glimpse of it is caught at some point on the line of cleavage between the blind and the seeing retina. This phenomenon we have called angular refixation. It is obviously a combination of pseudorefixation and real refixation. Again, if the patient has refixed several times from a certain fixation point on the blind side, and then instead of returning to the fixation point we use a light still farther out on the blind side, the patient's eye will often be noted to seek the center again before searching the blind field for the light. This we have called central pseudorefixation. This lighting of a light one step farther out on the blind field each time is very confusing to a patient, especially if we change the axis, and there is always at first a tendency to pseudocentral refixation, which in this series decreases on repetition. If after this sort of test we refix the eye on the center and then turn the fixation light out, we may often note, particularly with the more intelligent patients, what we have called angular pseudorefixation—that is, the eye is seen to move out along some axis to the point of a previous light, and then at an angle still farther out and on another axis, to the point of another previous light. A very intelligent patient usually at about this stage develops a seeking method of beating the game by a systematic search each time the light disappears, in one of two ways—either by moving the eye quickly on the horizontal axis until the light is or is

not discovered above or below, or by a circular motion sweeping the periphery of the blind field each time it is necessary.

It is notable in these cases that the reaction times in anterior lesions, producing homonymous hemianopsia or blindness in one eye and hemianopsia in the other, are of very much the same degree of magnitude as found in posterior cases, provided mental conditions and central vision are much alike in the two groups. But the reaction times in anterior lesions producing bitemporal hemianopsia may be somewhat greater at first than in lesions, either anterior or posterior, producing homonymous hemianopsia.

In explanation of these findings, however, several factors, independent of the supposed reflex arc, must be considered. Posterior cases have usually acquired rapidly a rather sharp homonymous hemianopsia, but with retention of good central vision. These cases have every incentive to learn rapid refixation; objects moving into the blind field may be plainly seen and followed. Thus the patient naturally develops the habit of looking on the blind side for objects which have disappeared. This ability of refixation apparently develops according to the mental ability of the patient. The only anterior lesions which could be fairly compared with this group are the rather rare cases of tract lesions giving homonymous defects, and the hypophysial lesions producing blindness in one eye and temporal hemianopsia in the other. Even in these two groups the central vision and the patient's mentality must be on a par with the central vision and mentality in the posterior case to which it is to be compared.

Hypophysial cases having a bitemporal hemianopsia form by far the largest group of anterior lesions. These cases, in binocular vision, have each defective field largely supplied by the good nasal field of the other eye. They have then an effective field of vision extending about 60 degrees from the fixation point in all directions. Accordingly they do not ordinarily observe moving objects disappear on passing to one side of the fixation, and therefore acquire no experience or habit of refixation. It is only in case vision in one eye is entirely lost that they gain such experience. Even with a large central scotoma in one eye, they do not become comparable to cases with complete homonymous hemianopsia, although they do acquire a certain amount of refixation ability. In addition to these considerations it must be remembered that hypophysial

cases are suffering from a glandular disorder which produces not infrequently a mental as well as physical weariness. But even with all these handicaps, cases of bitemporal hemianopsia commonly reduce their reaction times markedly in the second stage of the examination, until they compare very favorably with homonymous cases. Further, it may be noted that even in these cases the influence of observation and experience, such as is obtained from the examination of the first eye, is shown in the examination of the second eye, as a tendency, on the first test, to search in the wrong direction for the light that has disappeared. That is, the second eye is moved in the same direction as the first eye until the patient becomes aware or thinks of the different relation of blind and seeing field in the second eye.

When we compare posterior cases of homonymous hemianopsia with anterior cases of homonymous hemianopsia (tract) and with hypophysial cases blind in one eye and hemianopic in the other, we find that their reaction times compare very favorably in all respects, even if no great allowance is made for the presence of central scotomas and mental conditions.

After studying the time and character of the reactions in all cases examined, we believe that while the test may be of value to a psychiatrist as a measure of a certain mental capacity, it is of no diagnostic importance in the localization of brain lesions. We now feel certain that phenomena which we have called pseudorefixation, angular refixation, pseudo-angular refixation, the tendency to central refixation, and pseudocentral refixation, and the marked seeking character of all refixation on the blind side, in all cases indicate the functional result of thought and memory processes rather than a reflex action. It seems to me quite possible that this test could be made available for the armamentarium of the psychiatrist. An instrument as elaborate as the one herein presented would not necessarily be required. One with smaller lights, no larger than an ordinary hand perimeter, could be constructed for portable purposes. One central light and two or even one light on the arms, all movable, could be operated by a battery handle and finger switch so as to give essentially the same tests as herein described. From this standpoint possibly the psychiatrist would be able to give a better idea of the region of the lesion than would the ophthalmologist. Certainly we hope this may be the case.

CONCLUSIONS

The distribution of field defects in anterior and posterior lesions encourages psychological factors, which greatly complicate the Wilbrand test.

The tendency to decrease the reaction time on repetition; the presence of seeking movements of the eye at all times during refixation; the presence of pseudorefixation, angular refixation, pseudo-angular refixation, the tendency to central refixation, and pseudocentral refixation, all invalidate the Wilbrand test as a topical diagnostic reflex.

The results obtained by the Wilbrand test are surely a measure of certain processes of observation, memory, and thought, and are not an indication of the integrity of a definite reflex arc.

The possibility that this method of examination may be useful to the psychiatrist and thereby, perhaps, contribute to topical diagnosis, is suggested.

I am deeply indebted to Prof. Harvey Cushing, not only for kindly facilitating this investigation, but also for generously allowing me the privilege of presenting this particular phase of his cases.

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TRAUMATIC PULSATING EXOPHTHALMOS

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Traumatic pulsating exophthalmos is of sufficient rarity to warrant the presentation of this case.

Mr. J. R. L., aged 39, May 31, 1913, was riding on a large open street car which was derailed against a tree and he was thrown to the ground, striking his head. After immediate care in the Mercy Hospital, Pittsfield, his physician, Dr. A. E. England, who has kindly given me his report, found him the next day dazed, with all signs of true fracture of the base of the skull; bleeding from the nose, ears and mouth, also vomiting. No paralysis of the face, all reflexes normal. The mind cleared in about four days and memory was gradually regained. Hearing on left side lost and there was slight ptosis of the left upper eyelid with constant complaint of dizziness and headache. On July 6, about five days after the accident, the patient "felt something like electricity" at the upper corner of the left orbit, which was found to be a definite pulsation and bruit. At that time there was marked diplopia, dilated pupil and beginning proptosis.

The patient, when sent to me on May 7, 1914, by Dr. Leo H. Neuman, complained of a constant noise in his head and a terrible headache.

The right eye vision 20/30, Jaeger 1. Eyeball decidedly proptosed, 30 mm. by Hertel's exophthalmometer, with limitation of outward motion. The veins of the lids, especially those at the inner corner of the upper one, prominent and tortuous. Conjunctiva of the lower half of the ball and the lower lid congested with many full dilated and tortuous veins, especially one in the lower culdesac which extends from the nasal side to within 6 mm. of the temporal margin. Pupil 3.5 mm. active to light and accommodation, tension normal, media clear, disk slightly oval (axis 90), sharply outlined with small central excavation. Both retinal arteries and veins overfull, especially the latter; no hemorrhages, although the disk congested. No palpable pulsation of the eyeball.

The left eye vision 20/30, Jaeger 2, the proptosis much more marked than on the right side, 40 mm. by the Hertel instrument. An extremely large mass of tortuous dilated blood-vessels on the upper lid. In the upper inner corner of the orbit is a large dilated vessel about 2 cm. in diameter transmitting a distinct bruit. Visible pulsation of the globe. Entire conjunctiva congested with numerous dilated veins of bright-red color, greatest over the inner half of the globe. Pupil 3.5 mm., active to light

and accommodation, tension normal, disk round, extremely full, tortuous, constricted, and distended retinal veins; occasional pulsation of retinal arteries, no hemorrhage or exudate. Limitation in field of motion, eye turned in 30 degrees, abducens paral-



Fig. 1.—Full face—definite mass in inner-upper corner of left orbit. Congestion of both eyeballs, full veins on both upper eyelids. May.

ysis. Bruit most intense over the left supra-orbital ridge although felt over the entire head. A systolic blowing murmur heard over the same area is stopped by carotid pressure.

In left ear a thin, gray retracted membrane. Hearing so reduced that only the high notes are heard at 2 cm. Large perforation of the cartilaginous septum of the nose.

Dr. Neuman in his complete examination found the apex beat in the sixth space, systolic pressure 120, diastolic 82.



Fig. 2.—View of vein fullness which outlines the palpable separation between the frontals; also shows the skin congestion.

On May 29, 1914, the first photographs were taken. The right eye was prominent, vision 20/30, Jaeger 2, the vessels on the upper eyelid tortuous and the bulbar conjunctiva congested. The left eye vision 20/50, Jaeger 6, showed a greater dilata-

tion of the ophthalmic vein, eyelid vessels overdistended, bright-red congestion of the entire eyeball, extreme exophthalmos visibly pulsating. By tropometer moves 15 degrees down, 10 degrees up, 30 degrees to right, 0 degrees to left.

Roentgen-ray examination failed to show any fracture, although there was very evident separation through the frontal



Fig. 3.—Left profile, showing tortuous vessels on the upper eyelid, the proptosis and the congestion of the bulbar conjunctiva.

bone extending from deep in the left orbit to the vertex between the parietals. The course of this depression is outlined by the large ophthalmic vein shown in photograph No. 1.

Wassermann and Noguchi tests negative.

Slight contraction of the field of vision for form as seen by the chart.

Pressure on the right globe reduces the ball, which then pulsates; the same pressure on the left causes a partial replacement with very strong pulsation and the patient complains of dizziness.

Carotid pressure or pressure on the ophthalmic vein deep in the left orbit stops the pulsation and bruit. Change of position has no effect on the exophthalmos, although lying down increases headache.

On Oct. 4, 1914, another photograph was taken which, if studied with No. 1, shows the changes which have taken place since May 29. The bulging vessel is decidedly larger, being 4 by $1\frac{3}{4}$ cm. in its widest part, over the supra-orbital ridge.

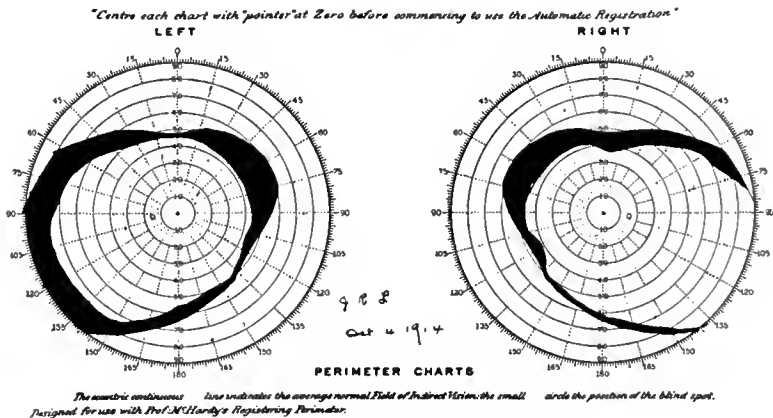


Fig. 4.—Field for form, Oct. 4, 1914.

A new venous dilatation is now seen on the right side over the lacrimal sac. This 2 by 1 cm. pulsating area was not present until this date.

October 4, O. D. vision, 20/20, Jaeger 1. Bulges 29 mm. by the Hertel instrument. Pupil 3.5 mm., no further change in the fundus. O. S. vision, 20/100, Jaeger 6, 37 mm. by Hertel. Globe directed forward and inward, but not depressed. Disk margin above and below somewhat blurred.

Close examination of photograph No. 3 shows the margin of a definite dull congestion of the skin starting on the right side of the nose, gradually widening over the forehead until it is about 7 cm. at its widest part. There has been no improvement in the general condition of the patient; the diplopia is unaltered, but as yet he will not consent to operation.

In 1907 deSchweinitz and Holloway published a monograph of great value, considering spontaneous and traumatic exophthalmos up to July of that year. The following brief summary

covers the cases of traumatic exophthalmos recorded since their article and also refers to three spontaneous cases, only one of which, however, did not appear in their report.

Yvert refers to Gasparrini's case reported in June, 1906, and included in the previously published reports, but again cited because of the treatment. Cases of exophthalmos are reducible by compression with pulsation murmur. The symptoms disappear on compression of the carotid, administration of potassium iodid and instillation of adrenalin for ten days.

Beauvois reports two cases, the first that of a woman of 41 who fell down stairs fracturing the base of her skull. A month afterward appeared right-sided exophthalmos without pain, and systolic murmur. Later occurred right-sided abducens paralysis, diminished vision and exophthalmos. Ocular and carotid compression was performed without result. Ligature of the common carotid three months after accident produced cessation of the murmur, but there was slight aphasia and left-lid paresis. The exophthalmos persisted, although somewhat lessened with gradual loss of vision. Ten days later the murmur reappeared, which could, however, be stopped by compression of the left carotid. Five c.c. of 1 per cent. gelatinized serum were injected into the thigh every other day. After three injections the murmur disappeared. On increasing the injection to 20 c.c., then to 50 c.c. and at last to 100 c.c., the exophthalmos disappeared, but paralysis of both abducens and slight dilatation of the pupil persisted. Three months after beginning of treatment there was a local cure.

Second case, a woman 53 years of age, had received a severe blow beneath the right eye with the handle of a pump, ten years before. First seen December, 1905. In August, 1905, intense pain in the right side of head. At the end of December, 1905, diplopia, exophthalmos, conjunctival reddening, systolic murmur. Fundus slightly congested. Vision right eye, 0.4, left eye, 0.6, paralysis of the right abducens. Visual field slightly diminished. Injection of $2\frac{1}{2}$ per cent. gelatin every five days in the thigh. After the twenty-second injection exophthalmos reduced one-half with normal motion and complete disappearance of the diplopia.

Santos Fernandez saw a man of 47, Dec. 16, 1905. Forty-two days before he had been struck by a hammer, which rendered him unconscious for three days. On regaining consciousness noise in head with moderate exophthalmos and ptosis.

Pupil normal. Conjunctiva congested. Compression of the carotid caused disappearance of symptoms. On December 24, started injection of 2 per cent. solution of gelatin and 2 per cent. of sodium chlorid; by January 20 much better. Pulsation stopped and exophthalmos disappeared.

Becker cites the case of a soldier who was wounded by the explosion of his gun on May 26, 1906. Four wounds on the right side of face, two on nose, one at the corner of his mouth and the other in the right eye. On the tenth day, protrusion of the left eye, which increased steadily in the following days. Retina showed hemorrhages and the movements of the eyes became more and more limited until entirely lost. On the eighteenth day pulsation appeared. Compression of the carotid caused diminution of the pulsation. Skiagraph showed a fragment of bone 2 by 1.5 cm. in the neighborhood of the cella turcica and in the cavernous sinus. July 3, 1906, ligature of the internal carotid. Relief for four weeks and then return of the former symptoms, but in eight weeks great improvement. After three months, further improvement. Vision with a +4, 5/15. Lateral movements of the eyeball normal.

Jacques reports a man of 25 who was injured in a motorcycle accident by falling against the wheel striking on the right side of head, fracturing the base of the skull. Three weeks later pulsating exophthalmos. Intense epistaxis nine times in six weeks from the right nose, which occurred after any strong effort. Ligature of the right common carotid. Three days later improvement, but after a short time recurrence of all symptoms. Still later, operative exposure of the inner portion of the orbit. A polyp was found in the sphenoidal sinus, which communicated with the cavernous sinus. Sphenoidal sinus packed with iodoform gauze, which was removed on the sixth day without hemorrhage.

Pooley reports a negro of 30, struck on the back of the head by a "spliced blackjack." Three wounds on the scalp, one on the parietal bone near its junction with the occipital, one further forward and another on the temple. He was felled by blow and rendered unconscious. On regaining consciousness, he was greatly disturbed by a most distressing noise in the head. Two days later all symptoms of arteriovenous aneurism present except pulsation. Eye protruded, blood-vessels of the conjunctiva and lids dilated. No pulsation of the eyeball detected, but when the ear was applied over the globe, blowing murmurs

and a continuous whirring, rumbling sound were heard. The eye could be pushed back into the orbit by the hand. Compression of the carotid diminished but did not stop the bruit. No change in the fundus and vision seemed unimpaired. Patient too illiterate for exact vision. Treated for a few days by a pressure bandage and compression of the common carotid. On March 16, ptosis, exophthalmos, enormous dilatation of the vessels of the conjunctiva and lids, pulsation easily obtained, especially near the inner canthus, papillitis, with enormous dilatation of the retinal vessels. One vein in the lower temporal region seemed to be obstructed and above and below the obstructed portion was greatly distended. Noises in the head had become more aggravated and to this was added occasional violent pain in the orbit. There was no impairment of hearing. Patient would not consent to operation.

In discussion of Dr. Pooley's paper, Dr. Knapp stated that he had had three cases, in all of which the ligation of the common carotid had relieved the bruit, exophthalmos and chemosis but the optic nerve went on to atrophy.

Oppenheimer had seen a case some years ago in which the carotid had been ligated. Secondary hemorrhage with fatal result had followed. It proved to be an erectile tumor.

Claiborne had seen a case ten years ago like Dr. Pooley's. He had applied pressure bandage, which was kept up for eighteen months, and had administered mixed treatment internally. A perfect cure resulted.

Gruening had seen the vision immediately restored after ligation of the carotid in a case operated on some years ago without anesthesia.

Tyson has seen atrophy follow the tying of the common carotid, although the other symptoms had subsided.

Flemming and Johnson saw a male of 47, September, 1907, who, although previously in good health, fell striking the back of his head and was unconscious a few minutes. A month later left hemianopia, no proptosis, but soon afterward constant beating noise in the head. April 19, 1908, left eye markedly proptosed, lids slightly swollen and congested, conjunctiva deeply congested with chemosis, especially in the lower fornix. Pupils equal, active to light and accommodation. Distinct pulsation of the left eye with limitation of all its movements. Marked continuous murmur with systolic exacerbations over the left eye and skull. Pressure on common carotid stopped murmur. Right

eye vision, 6/12; left eye vision, 6/24. Right half each field blind. Fundi normal. May 14, left common carotid tied at the level of cricoid cartilage, pulsation stopped.

Barrett and Orr cite the case of a male aged 50 years who, ten weeks before while intoxicated, fell and remained unconscious from twenty-four to thirty-six hours. In hospital for three weeks. No evidence of organic lesion. Six weeks after the injury he complained of buzzing in the ears and a few days later his right eye was prominent, with vision 6/36. The lower lid everted, marked exophthalmos in the middle line, conjunctiva of the lower lid engorged and much swollen, fundus veins dilated. The external canthus was divided and the orbit explored, the finger being pushed behind the eye, where a pulsating mass was felt. March 2, a marked bruit over the temporal bone and eye, synchronous with the pulse. March 4, vision as before but eye was almost stationary, with many hemorrhages and distended veins in the fundus. Neither rest nor medical treatment stayed the progress, so the common carotid artery was ligated March 11. Steady improvement, exophthalmos diminished until comparatively slight at the last examination. The movements of the eye returned in part, the action of the external rectus being most limited and the hemorrhages disappeared from the fundus. Bruit lost after the operation, returned about a fortnight later. Then gradually disappeared again. In October there was slight proptosis. Vision, 6/12.

Barbieri tells of an Italian, male, 43 years old, who reported on April 24, 1907, with an intense pain in the temporal region. On the succeeding day the left eye became prominent and a month later the right eye pulsated. Injection of 5 per cent. gelatin tried without result. October 21, ligature of the right common carotid. The exophthalmos and pulsation continued. On Jan. 8, 1908, the left common carotid was tied. On April 4, the day of his last examination, the exophthalmos continued, the movements of the ball moderately free, pupil inactive to light, headache slight, protrusion and subjective noises persisted.

C. S. Merrill in a personal communication reports a Miss E. T., aged 50, who, on July 5, 1909, fell from a wagon, striking the back of her head. When seen there was much chemosis on left side with extreme pulsating exophthalmos and aneurysmal bruit. Compression of the carotid stopped the bruit. Vision right, normal; left, nil. Dr. A. W. Elting ligated the left external carotid. All symptoms were relieved and the eye-

ball returned to proper position. Some months later vision in right eye with correction was 20/20. Left vision, objects to the outer side, optic atrophy.

The wheel of a loaded wagon passed over the head, just back of the eye, of a twenty-four-year-old man, Jan. 29, 1909. Halstead and Bender report that he was unconscious three days. Left eye bulged more at the time of accident than it did when he entered the infirmary, when it was turned sharply toward the nose and appeared paralyzed. The lids were swollen and it was impossible to close the upper lid, move the jaw, the tongue or to swallow. Roaring noises synchronous with the heart beat could be heard from the time patient regained consciousness until he entered hospital. He remained in bed for two weeks following accident, during which time the sight in his left eye became much impaired. Examination made on Aug. 27, 1909. The left eye turned in 45 degrees and was very prominent, $\frac{2}{3}$ of an inch farther forward than the right. Veins on the outside of the upper lid were very large and tortuous. The blood-vessels of the sclera were also dilated. Cornea vascular and over 4 mm. square area in its center there was a fairly dense cicatrix. Vision right eye 20/30-3, left eye 20/120. September 28, vision right eye 20/20, left eye 20/50. On October 10, a pulsating tumor felt over the lid and in the inner angle of the orbit; bruit heard over the left temporal region and patient complained of constant roaring noises, heard most distinctly in the left ear. On October 14, left internal carotid ligated near its origin with chromicized catgut. At present, the left eye turns inward and there is some exophthalmos but no bruit.

Lane's patient was struck on head with a brick three months before, fracturing his nose. Twelve days later ectropion lower lids, marked edema. Vision 8/200. Right eye, enormous dilatation retinal veins, small hemorrhages about disk. Pulsation, bruit, exophthalmos. Blowing sound left ear. Pulsation continues over left temporal region with accentuated systolic murmur. Only general treatment and K I. On June 10, patient sat up for first time. At 5 a. m., 11th, aphasic with right facial paralysis. On September 2, no pulsation, faint systolic murmur, conjunctival vessels bright-red color. Vision left eye 6/6.

Orloff's pulsating exophthalmos developed six months after a deep wound in the region of the left parietal and temporal

bones in a 30-year-old patient. A successful result followed ligation of the ophthalmic vein in the depths of the orbit, after a temporary resection of the external wall of the latter.

Friedenwald's case of a colored woman of 20, although not of traumatic origin, is recorded because of the absence of bruit.

Halstead reports a man of 36, who was struck twice on the left temporomalar region with a hard instrument, Feb. 20, 1910. Unconscious a short time then walked home, blood from ears and nose. Facial paralysis next day and roaring in the ears. Two months later, when the left eye was enucleated for panophthalmitis, orbital contents prolapsed. Vision right eye, 20/40. Diagnosed as pulsating tumor base of brain, probably aneurysm. Oct. 5, 1910, stump of left eye removed. Right eye signs of beginning venous stasis. Roaring in ears heard through head, long, loud bruit over malar regions. Common carotid and two divisions of superior thyroid ligated. Immediate disappearance of bruit. No brain symptoms.

Zeller's patient, 35 years of age, on Dec. 24, 1894, shot himself in the right temple beneath the zygoma and in front of the ear. Unconscious. Three weeks after the injury exophthalmos with ecchymosis on the right side. Jan. 24, 1895, exploration of the right orbit. Vision 1/3. Movements of the ball limited. Globe directed downward and inward with abducens paralysis. March 5, 1895, exploration on the site of old operation of the neck. Carotid removed between ligatures with no improvement. On April 13, 1895, resection of the frontal bone opening the orbit. An attempt to tie the cavernous and carotid sinus within the orbit resulted in intense hemorrhage and death in eight hours. Necropsy showed that part of the bullet had engaged almost at the apex of the petrous portion of the temporal bone having entered the internal carotid and the cavernous sinus.

Kraupa, in 1894, saw a 20-year-old man who, while in bathing, dove from a dock 2 meters in height and before he came to the top of the water felt an intense sound in the left ear which in the next few days constantly increased. Typical pulsating exophthalmos of the left eye. No operation.

McClellan tells of a man of 32 who was injured eight months before. A machine at which he was working upset, striking him on his chin. Unconscious twenty-four hours, with considerable hemorrhage from both the mouth and nose, but none from the ears. Slight exophthalmos developed three months later,

which grew steadily worse until both eyes were decidedly prominent, especially the right. A distinct pulsation, rhythmical with the pulse, could be seen and felt, also more on the right eye. The conjunctiva was very edematous and the veins very markedly dilated. Movements of the eyes and visual fields for form normal, but it was impossible to obtain the color fields. Vision, right eye 20/20-3, left eye 20/30-3. Bruit audible over the bifurcation of the right common carotid and reached its maximum intensity about 1 inch behind the external angular process of the right frontal bone; distinctly audible over the left side, but not so marked and of a different character and higher pitch. By compressing the right common carotid the bruit and the pulsation of the eyes stopped. Compression of the left carotid had no effect. Vision reduced to 20/50 in each eye. Ligation right internal carotid with immediate cessation of the bruit and pulsation of the eyes. No return. In one week the engorgement of the retinal veins and choked disk disappeared and vision returned to normal, although the conjunctiva still congested and the exophthalmos only slightly reduced.

Lystad's 15-year-old stoker was shot with a revolver bullet through the right nose in August, 1902. Spastic hemiparesis with incomplete anesthesia. Three months in hospital. Resumed work nine months later. Two years later a swelling over the right eye with exophthalmos and a systolic murmur made its appearance. Five years after this accident, he was struck with a fist, when he again came to the clinic. Eye movements then normal. Pupil slightly small. No pulsation. Subjective and objective disturbances completely lost on digital compression of the right carotid. Sept. 17, 1907, ligature of the internal carotid and the external carotid and jugular veins. Temporary improvement. After a time pulsation and exophthalmos returned. In November, 1907, electrical treatment considered worthless. In beginning of 1908, condition about the same as before the operation. Patient was unable to work. Jan. 24, 1908, orbital operation by Schiotz. After three weeks an objective improvement, but still an intense headache and slow pulse. In the following week swelling became less and pulse normal. Out of bed for the first time in two months. Vision, fingers at 2 meters, visual field contracted, tension plus. After a few months the patient was able to work again and the protrusion was hardly noticeable. The glaucoma, however, increased, miotics having no effect. Conservative operation not permitted by patient. Eye

was enucleated in December, 1908, after which he had no headache and the murmurs were great only when lying down.

Risley reports two cases. The first patient was knocked unconscious by a fist blow on the ramus of the right jaw. Four weeks later pain in the head, diplopia and confusion. Proptosis of right eyeball about 10 mm., swelling of the lid, conjunctiva chemotic with full veins near the inner canthus. Limitation in motion, excepting downward. Systolic pulsation best heard over the right eye. Vision 6/12.

The second case was a male of 30 who, three years before, had had his head caught between a trolley car and an express wagon. Unconscious seven weeks. Abducens paralysis, slight proptosis of the right eye. Loud blowing, systolic bruit heard over the entire skull but loudest over the right eyeball and left frontal. Bruit lost on right carotid pressure. Roentgen ray negative. Both cases are still under observation.

A man 18 years of age, who had been struck on the head with a piece of iron, was seen by Ginzburg. He was unconscious fifteen minutes with bleeding from the mouth and nose. On examination, hemorrhage in the left lower lid; right lid swollen, ptosis, marked injection and protrusion of the fixed globe and definite systolic pulsation. December 7, ligature of the right common carotid. No change, so March 27, 1911, clamping of all vessels within orbit by forceps, which were left in the wound. March 30, protrusion of the eyeball greater than before the operation. July 17, protrusion very much less. Aug. 10, 1911, ptosis slight, thickening of the bulbar conjunctiva. Protrusion entirely disappeared. Eyeball freely movable. No conjunctival hyperemia, no pulsation. Vision right eye, 0. Vision left eye, 0.5.

Savariaud examined a small girl who, after having had her head squeezed between a large plank and a boat, had a hemorrhage from the right ear and nose. Later paralysis of the abducens on the right side, exophthalmos and a continuous murmur with systolic emphasis. Slight pulsation. Injections of gelatin after Lancereaux's method failed.

James and Fedden's first patient fell 9 feet striking on his head, became unconscious, but only had a scalp wound, some bruising over the right frontoparietal region, with bleeding from the nose and a double black eye. The right pupil was semi-dilated and sluggish in its reaction to light, while the reaction on the left side was brisk. On the next day there was very extensive ecchymosis on both sides, that on the left being the

more marked. Ten days later left-sided ptosis and a pulsating exophthalmos. Treated by a rest in bed and a pressure bandage. Three weeks later a commencing keratitis à lagophthalmos, so the lids were sewed together. Three days later the left common carotid was tied. After a couple of months no exophthalmos or pulsation in the orbit and when the lids were separated the left palpebral fissure was slightly smaller, the left pupil contracted, the vision 6/18 and that of the right, 6/6.

Second case, woman aged 53, four and a half weeks before had fallen down some steps and became unconscious. Small wound over the right external angular process. Kept in bed for three weeks, but when she began to go about complained of right-sided headache and the right eye began to swell. A normal recovery from her initial head injury; for the first few days there had been considerable subconjunctival ecchymosis, which had rapidly absorbed. No ocular palsy. Pupillary reactions normal until she got up, when there was right-sided ptosis with proptosis, moderate edema of the ocular conjunctiva, with dilated veins. Eye stationary, the pupil dilated and fixed. Vision, fingers 6 feet. Very marked venous pulsation of the disk, which seemed to jump forward at each beat. No pulsation of the globe. Put to bed with a pressure bandage and given iodids internally. Two days later definite pulsation in the orbit and a faint systolic bruit over the right temporal fossa. Proptosis increased with headache and buzzing noise in the head. Bruit plainly heard over the eye, forehead, vertex and temporal fossa. The right internal carotid tied just above the bifurcation stopped pulsation. Five weeks later patient discharged, slight proptosis, very slight edema of the conjunctiva with fulness of the veins and marked pulsation of the disk. When last seen, ten months later, eye moderately convergent, abducens paralysis. Upward and downward movement fair, vision right eye, Hm. 1:5, 6/18; left eye, Hm. 1:5, 6/6. The external recti remained paralyzed and the disk pale.

Buchtel operated Aug. 13, 1912, on a patient who was struck on the head with a pitchfork three months before. He had a buzzing sound in his head and in a few days the left eye protruded, well marked exophthalmos forward and downward. Pulsation of the eyeball and the mass at the upper-inner angle of the orbit with a continuous bruit and systolic accentuation over the brow and temple. Complained of the noise, diplopia and headache. Fundus negative. Operation: incision eyebrow,

angular, superficial and temporal veins tied and superior ophthalmic ligated as far as possible in the orbit. More edema at first but no bruit, pulsation or fundus change. Exophthalmos gradually lessened. Vision, 20/20.

Buchtel also reports two other unrecorded cases, one a double exophthalmos cured by ligation of the common carotid; the other unilateral, died after the same operation.

Matthewson saw a man of 32 who had been thrown from top of car Oct. 8, 1910, fracturing the base of the skull. November 5, left eye very much proptosed with complete ptosis. Considerable swelling of the conjunctiva and much restricted motion of the globe. Fundus negative. Vision, fingers 8 feet in the upper field, the lower field lost, no pulsation of the globe. December 20, veins upper lid much dilated, ball pulsated, slight nerve pallor, slight dilatation of retinal veins, no bruit, occasional headache. March, 1911, vision almost gone. In August condition more marked. Loud blowing murmur, left temporal heard over greater part of skull. September 5, common carotid artery tied. Month later little proptosis, no bruit or pulsation.

Cunningham's case was that of a man of 39, who four years before received a severe blow on the left cheek, that night felt bad, spat blood. Two days later roaring in his head began, gradually worse. Two months later sudden pain right temple with marked protrusion of eyeball. Right common carotid clamped. Bruit and pulsation lessened at once, lost in four days, although two months later exophthalmos still present. Later clamp removed by operation.

Maher saw a man of 35 who, ten months before, had had a blow on his head with a stick, was unconscious thirty hours and dazed a week. Three weeks after injury right eye began to protrude and throb with marked pulsating exophthalmos, controlled by pressure on the right common carotid. Bruit over eye and temporal region. Marked engorgement conjunctival vessels, edema conjunctiva; pupil dilated, retinal veins dilated and tortuous, marked edema of optic disk and retina. Headache and whirring noise in head. Right internal carotid tied. Pulsation ceased at once. Exophthalmos diminished. Three years later no exophthalmos, slight disk pallor and only occasional headache.

The second patient, a man of 19, fell 45 feet, fracturing the base of the skull. Unconscious several days. Six weeks after injury left eye began to protrude and for some weeks progressed

then gradually subsided, the right eye became more prominent. Pain and noises in the head. Right eye proptosed and convergent, conjunctiva edematous, vessels full, limited motion of the globe up and down, no motion outward, pupil normal, retinal vessels slightly tortuous and dilated. Vision 5/18, Jaeger 1. Left eye not proptosed, movements upward and downward limited. Pupil dilated. Disk pale, vision 5/21, Jaeger 2. Loud bruit over right eye and temple. Left internal carotid tied. One month later marked subsidence, eye moved up and down but not out, headache better but bruit persisted, vision same.

DeSchweinitz and Holloway collected 54 cases of traumatic pulsating exophthalmos, making a total of 214 on record in 1907. Since then 32 others have been recorded, making a grand total of 246.

In a summary made from the 32 here correlated it is found that the common carotid has been ligated fourteen times, resulting in cure in 4, improvement in 6, failure in 4; internal carotid, cure in 2, improvement in 4. One case was later cured by the removal of a polyp in the sphenoid sinus, extending from the cavernous sinus. Orbital operation has resulted in 3 cures and 1 failure; gelatin cure in 1, improvement in 1, failure in 1; no operation as yet reported in 5; one patient died after an attempt to tie the cavernous sinus after ligation of the common carotid had failed.

It is impossible to draw conclusions from the cases here detailed, partly because of the small number and partly because the mere citation of treatment does not explain the actual condition demanding the operation. Ligation of the common carotid is the operation most often performed, but as it may cause death, many fatalities having been recorded, the other operations should receive greater attention in the future. After a careful study it seems that ligation of the superior ophthalmic vein is an operation that should always be done after ligation of one common carotid has failed and in many cases may be advised as the primary procedure. Although three cases have been cured by the injection of gelatin, it is not here recommended, nor is the treatment by drugs, rest or even the compression of the carotid.

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DISCUSSION

DR. T. E. OERTEL, Augusta, Ga.: In discussing this paper, I wish to present the following case:

H. Z., aged 39, railroad engineer, was injured June 21, 1901, in a railroad wreck. He was unconscious four days. Hemorrhage from nose and ears. Diagnosis, fracture of the base of the skull.

Previous History: I saw him first August 10, two months after the injury, at which time he gave the following history: Within a day after the accident the left eye began to inflame and protrude. About one month later the right eye began to follow the same course. There had been intense pain, diminution of vision, photophobia and laceration. His physician had kept his eyes bandaged in an endeavor to reduce the exophthalmia. After some days the eyes both became nearly normal. July 23 he suffered another attack, which this time began in the right eye. Five days later the left eye developed symptoms and followed a course similar to that of the right.

The patient was confined to bed. Both eyes presented intense chemosis, the lids could not be closed on account of this and the highgrade of exophthalmos; the pupils were dilated and immobile and the globes fixed on account of the swollen condition of surrounding structures. Distinct pulsation of both globes could be seen and felt, while across the bridge of the nose a small vessel could be felt to pulsate and presented a marked bruit. Vision was reduced to counting fingers at two feet. Pain was a prominent symptom.

Treatment consisted in the internal administration of iodids and sedatives, locally in scarification of the chemotic conjunctivae, leeches to the temples and astringent washes. By August 20 the condition was much improved; September 15, vision, O. D. 20/15, O. S. 20/100; September 24, vision, O. D. 20/10, O. S. 20/30; December 13, vision, O. D. 20/20, O. S. 20/20. Chemosis and exophthalmos and pulsation have disappeared and the bruit and pulsation in the nasal vessel can no longer be perceived. There remains a total paralysis of both external recti.

The patient was soon after able to resume work as "hostler" engineer, which service he has continued until the present time. There has been no further recurrence of his exophthalmos but the external recti have never regained their power.

Doubtless the eye symptoms were caused by an arteriovenous aneurysm, due to a rupture of the internal carotid into the cavernous sinus. Spontaneous cure evidently resulted by obliteration of the sac with a thrombus.

The history of this case may point to conservatism in the treatment of like conditions. In other words, it would seem wise to give nature time to effect a cure before resorting to the serious expedient of ligation of the carotid. Serious cerebral symptoms arise in about 25 per cent. of ligations of this artery and about one-half of these cases terminate fatally.

We should therefore approach this operation with caution in any condition where life is not endangered and there is a reasonable assurance of spontaneous cure.

THE INTRANASAL PARTIAL RESECTION OF THE TEAR SAC

J. SHELDON CLARK, M.D.

FREEPORT, ILL.

The subject of blennorrhea of the nasal duct, with accompanying inflammation of the lacrimal sac, is one which should be more thought of by the medical profession as a whole. Time was when all we thought of doing for the alleviation of those suffering from this distressing and disfiguring affliction was the use of measures of more or less temporization. Repeated probings are of little value where there is a stenosis in the bony portion of the nasal duct, and it is here that the usual stenoses occur. In my opinion, where probing is to be thought of, if a result is not obtained in two or three sittings, one should desist in the use of the probe and make preparation to do something more radical. The wearing of copper wire stiles is objectionable to the patient in many ways, being a constant reminder of a physical imperfection.

Chronic dacryocystitis is a very dangerous affair for any one to temporize with. The trouble is that it is like the dynamiter's bomb or his bottle of nitroglycerin; it is likely to go off at any moment. One with a sac infection is liable to a corneal complication at any time; and if the conjunctival sac is continually bathed in pus, what a fine opportunity for infection to take place and cause a corneal ulceration on the slightest injury to that structure. Fuchs, in his text-book, says that acute dacryocystitis causes fully one-third of all the cases of *ulcus serpens*—that dreaded disease of the cornea which is so disastrous to eyesight.

To my mind, there has been entirely too much procrastination with regard to chronic tear-sac infections. We would not temporize so with pus sacs in other parts of the body, and why here. Up to the present time, the Meller operation for the total excision of the tear sac has given good results. There are objections, however, to this procedure, and I think that West, in his operation, has overcome these objections. One can operate by the intranasal route in cases of phlegmon. The external extirpation of the sac could not well be attempted in such a condition. Patients have a fear, in some cases rightly so,

of a resultant scar. Then there is a query regarding the disposition of the tears after the total removal of the sac. We can, with much assurance, tell the patient that there will be no scar, and yet scars, or at least cicatricial ridges, do sometimes follow. Then as to the tears. Although we can state that there is a marked diminution in the secretory function of the tear glands, all of which is more or less conjecture, yet we can say that in the event there are too many tears tumbling over the face, we can remove the tear glands. But all this does not make for the peace and comfort of mind of the prospective patient, and he is quite apt, as has been my experience, to delay the operation indefinitely. It has been my experience that the intranasal operation is more easily secured, especially in women, for whom the cosmetic element is of import. I think this is rightly so, for the patient operated on intranasally for the resection of the tear sac has a functioning tear apparatus and has neither the anticipation nor the realization of any external defect; furthermore, the tears go into the nose as they anatomically should.

Parsons (*Pathology of the Eye*, vol. ii, p. 750) says: "The sac is lined by cylindrical epithelium, usually in two layers, the inner layer consisting of very high cells, 35 to 50 microns. There is a definite basement membrane. It is doubtful whether the cells ever bear cilia. There are often many goblet cells. Beneath the basement membrane is an adenoid layer of the usual type. It is doubtful if ever it contains follicles normally. The submucosa contains dense fibrous tissue which is very vascular, especially on the part adjacent to bone. There is no muscular coat, but elastic fibers are abundant."

In cases of stenosis, as a rule the point of the inflammation and the resultant stenosis occur at the narrowest point in the lumen of the duct, and this point is just beyond the lower end of the sac, at the beginning of the nasal duct. It is very easy to see, in turgescence of the duct, that at this point the epithelial cells are first encroached on, their blood supply interfered with and a resultant inflammation occurs.

Infections of the sac can be either ascending or descending. Rarely, if ever, has West found that suppuration in the ethmoid cells is accompanied with suppuration in the tear sac, although this structure is at times nearly surrounded by the anteriorly placed ethmoid cell.

One condition that exists more frequently than many suppose is tuberculosis of the tear sac. Bribak (in the clinic of

Professor Axenfeld, University of Freiburg) reports on the material in that clinic and states that in the past few years twenty-five cases of tuberculosis of the tear sac has been found, although only a part of the extirpated sacs were examined microscopically. In their experience, tuberculosis of the tear sac has been the only demonstrable lesion of tuberculosis found at necropsy. Axenfeld describes the cases of tuberculosis of the tear sac as being "doughy, elastic, and having slight resistance." It would seem, therefore, a matter of moment to make a diagnosis of tuberculosis of the tear sac before the process has reached beyond the tissues of the sac. Conservative treatment in these cases is only putting off the evil day.

My attention was first called to the operation of Dr. West by Professor Holth and his friend Dr. Heidenrich, both of whom had made the trip down from their homes in Christiania to Berlin for the purpose of studying West's technic.

At that time Dr. West had records of over two hundred cases that he had operated on for varying conditions of the tear sac. These included phlegmons, chronic dacryocystitis, dacryoblennorrhea, fistulae and secondary operations following the Meller excision procedure, the last on account of a persistent epiphora.

The operation is highly satisfactory in the conditions above mentioned. Before selecting a case for this operation one should examine the canaliculi to see if they are smooth and patent. If they are not, the results are correspondingly poor, since the canaliculi must be able to perform their function and get the tears over into the sac, or what is left of it, in order that you have a satisfactory or positive result. One punctum should work, preferably the lower one.

The relation of the ethmoid cells to the fossa lacrimalis is interesting for the fact that they vary so much in different skulls. Usually the ethmoid cells all lie posteriorly to the lacrimal fossa, and this is very interesting to note in an anteroposterior section. Occasionally one or two cells extend laterally and anteriorly, thus in a measure hindering one in getting at the sac. Again, large cells may lie in the tip of the middle turbinal and together almost completely cover the floor of the lacrimal fossa. However, these deviations from the normal are not unsurmountable, as we will later see.

The lacrimal fossa is formed by the junction of the paper plate of the lacrimal bone and the nasal process of the superior maxillary bone. There are three types of fossae: first, where

the maxillary bone forms by its nasal process two-thirds of the fossa, and the paper plate one-third; second, where the greater part of the floor of the fossa is composed of the nasal process of the superior maxillary bone; third, where it is mostly the thin paper plate of the lacrimal bone. It is therefore to be understood that in one case ready access is to be had to the sac and in another it will take considerable chiseling to break down the floor on account of the denser structure of the maxillary bone.

The principle of the operation is to cause the tears to pass through the sac directly into the nasal chamber, without descending the duct to the inferior meatus. By doing this we short-circuit the duct. The idea of getting drainage through the nose and thus effecting a cure in cases of dacryocystitis is an old one. The ancient Greeks and Egyptians had their ideas regarding the *modus operandi*, using everything from a hot poker up the gauntlet of the medieval surgical instrumentarium. Caldwell in 1893 reported a case as a cure in which he had run a sound from above downward to the point of stenosis and then burred up from the nose until he came to his sound. He was obliged to take away the anterior end of the inferior turbinate.

In 1899 Seifert of Würtemberg reported the removal of the tear sac for tuberculosis, and Professor Killian in the discussion mentioned that he had removed the anterior end of the inferior turbinate, thus getting to the duct for the treatment of that structure for tuberculosis.

In 1903 Passow, then at Heidelberg, reported four cases of the Killian procedure, in which he did a preliminary inferior turbinotomy, and then under general anesthesia attacked the duct. It will be seen that all these procedures called for the resection of the anterior end of the inferior turbinate body and the channeling up through the duct to the sac.

In 1908 West began his work and until May, 1910, had operated on seven cases. The only difference in his work and that of the others just preceding him, was that he attempted more conservatism. Among seven cases he found it possible in three to push the sound down the duct and dissect out the sound in the nose, above the inferior turbinate body, thus leaving that structure intact. In the other four cases this was impossible for him to do; for the reason that he could not pass a sound through into the nasal duct. Therefore, in these cases it was necessary for him to open the canal above the inferior turbinate and follow the canal up sufficiently high so that it was possible

for him to pass a sound horizontally through the canaliculus into the nose. He called attention to this operation in Washington and Berlin, and termed this the window resection of the nasal duct. In three of his cases he opened the sac as well as the duct. At this time he always dissected the sound loose in the nose and this procedure proved very difficult.

In 1910 West read a paper before the Berlin Laryngological Society, and the paper was published in *Fränkel's Archives für Laryngologie*, giving the technic of the operation as then done. In 1911 West returned to Berlin and again took up the work on the duct and sac operation. He then began to do nothing but the sac operation. It was very hard for him to get patients and up to July, 1912, he had done but forty cases, and a number of these were shown to the Ophthalmological Society of Berlin. He now took up the work in the Poliklinik of Professor P. Silex and was able to secure an unusually large number of cases, so that in February, 1913, West described his technic before the Laryngological Society of Berlin, with a report of over a hundred cases. In April of that year he gave a demonstration before the Berlin Medical Society and at that time had done 119 cases. Up to December, 1913, he had a record of over two hundred cases with a positive test present in over 90 per cent.

The technic of the operation is divided into four steps, a preliminary incision of the lower punctum having been made a day or so previous to the operation.

1. If the nasal septum is deflected in such a manner as to obstruct the view, then a partial resection of the septum is made. It is preferable to do this from the opposite side from that on which the tear-sac operation is to be performed.

2. When the anterior end of the middle turbinate body is large and covers the fossa, then it is necessary to resect the anterior portion of the middle turbinate.

3. It is necessary to make a mucous membrane flap with the view of not alone providing a window at the floor of the sac, but also to gain room so as to properly view the parts to be attacked. The lines for these incisions are shown in Fig. B.

4. Resection of the edge of the processus pyramidalis is made when this structure presses too much toward the median line. To do this, the bone must be denuded of its covering both internally and externally.

The work on the septum, turbinate and processus pyramidalis are necessary where there is a departure from the normal con-

figuration within the nasal cavities, and then only when there is much obstruction to a view of the parts.

A preliminary anesthesia is obtained with cocain and adrenalin and in the usual manner. It is well to inject a solution of a 0.5 per cent. solution of novocain at the junction of the skin and mucous membrane, and in the neighborhood of the aperture pyriformis.

The parallelogram of mucous membrane removed from the region of the fossa of the sac is placed in normal salt solution at body temperature, and thus is saved for use in covering the resected end of the middle turbinate body, provided this structure has been attacked, and not even then if there have been any ethmoid cells that have been opened, the reason being that it would prevent drainage.

The torus lacrimalis is the name given by West to a very important anatomical landmark that lies just underneath the sac. It is caused by the floor of the sac pushing itself toward the nasal chamber and thus gives rise to a protuberance, and it is by this structure that one locates the position of the sac. The chisel is then placed on this protuberance and directed as if intending to enter the eyeball at its equator. The work with the chisel is under the direct observance of the operator, while an assistant, properly trained, makes gentle taps on the chisel with a hammer. The operator can readily tell when he has broken through the bone. The bone forming the floor of the fossa is thus removed. On getting the sac clear of bone, sufficient to make the desired resection (and one should see to it that the floor of the fossa lacrimalis is removed well up under the sac), one should then orientate himself with a sound passed through the canaliculus, gently pressing the sac into the fenestra made in the bone; the sac is then grasped with grasping forceps, and having secured a firm hold on its wall, it is resected. One should endeavor to get away a good sized portion of the sac.

NOTE.—Since writing the above and for the past three months I have been using almost altogether my special punch illustrated in this paper and of which I speak under the heading of instruments.

THE AFTER TREATMENT

A small quantity of xeroform gauze is used for lightly packing the nose. Boric acid solution is gently irrigated through the canaliculus and sac until it appears in drops from the nose. On the third day, the packing having been removed, sterile water

is injected through the canaliculus to wash out all clots that have formed, repeating the irrigation every five minutes until the water comes out clear.

An operation is not considered successful unless the color test can be secured. This consists of the following procedure: A 2 per cent. solution of fluorescein is dropped into the conjunctival sac, and a pledget of cotton is placed under the middle turbinate body; in a few minutes the fluorescein will stain the cotton. To facilitate the downward flow of the fluorescein stain, have the patient exercise the lids violently.

CONCLUSIONS

The intranasal resection of the tear sac has many advantages, and they are almost all of them for the patient.

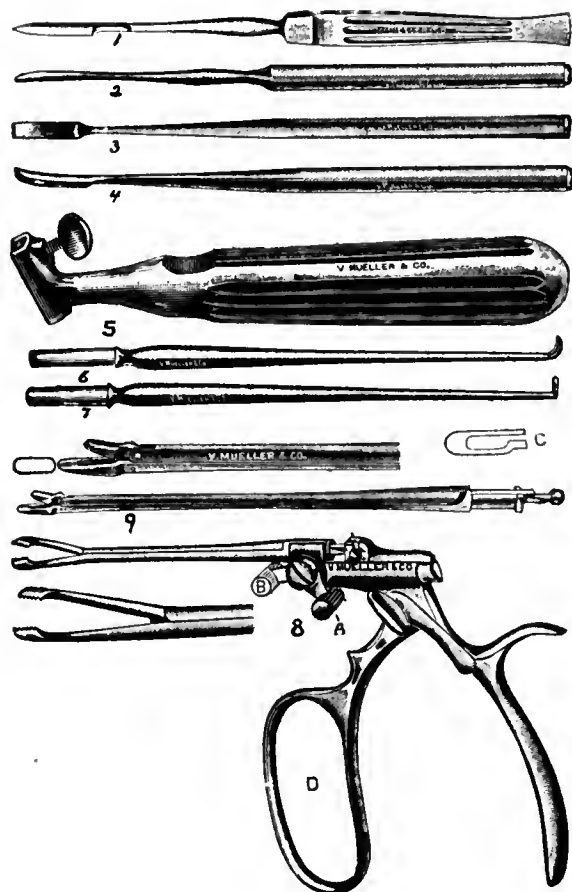
1. A functioning tear apparatus is the foremost attainment.
2. There is no possibility of an external scar, nor the dread of one.
3. There is no epiphora following the operation.
4. There is no possibility of having to do a secondary extirpation of the lacrimal gland on account of troublesome epiphora.
5. Epiphora due to stenosis is permanently cured.
6. The operation may be performed while a phlegmon is present and with utter unconcern.
7. Since the West technique was first mentioned by me in a paper read before the Illinois State Medical Society at its annual meeting held in Decatur, Illinois, in May, 1914, there have been several modifications offered. The proof of their efficacy will be the end results obtained. What we want is a cure of the tear sac pathology and have a functioning tear apparatus left for the patient.

Finally, I wish to thank Dr. J. M. West of Berlin, Germany, for his many kindnesses extended me while working with him in Berlin and the privilege he extended me of presenting this subject to my confrères at home.

INSTRUMENTS

To perform the operation as outlined it is well to have a few special instruments. West has devised these, and these with some added modifications may be obtained of the firm of V. Mueller & Company of Chicago. The grasping forceps as used in the universal handle obviates the necessity of a right and left. There is also a light punch forceps made, obviating the necessity of using the knife in the manner it is now used to

cut off the sac. This will be accomplished by making a slit in the lower end of the sac as it protrudes into the opening and then by pushing in the punch forceps, bite out a window in the sac. This forceps is necessarily light and is not intended to deal with bone, but simply with the membranous sac. For the other measures spoken of in connection with the sac operation proper,



there are sufficient instruments now on the market to accomplish the work:

1. A long handled knife that has a thin cutting blade.
2. A muco-periosteal elevator with a specially curved tip.
3. A straight chisel.
4. A chisel with a special curve so as to properly engage the torus lachrymalis in the initial attack upon the bone forming the lacrimal fossa.

5. Universal handle for use with the knives.
6. A beak knife for making the longitudinal incisions in the mucoperiosteum.
7. A right angled knife for the rather vertical incisions in the same structure.
8. Shows the special grasping forceps that may be used for right or left and is fitted in the universal handle. (There is a deflection near the beak of this forceps that enables one to use this instrument, and which is not shown in the illustration.)

In several of my cases I have found some difficulty in grasping the sac wall, it being a rather dense structure and tightly drawn across the opening made in the lacrimal fossa. The probe

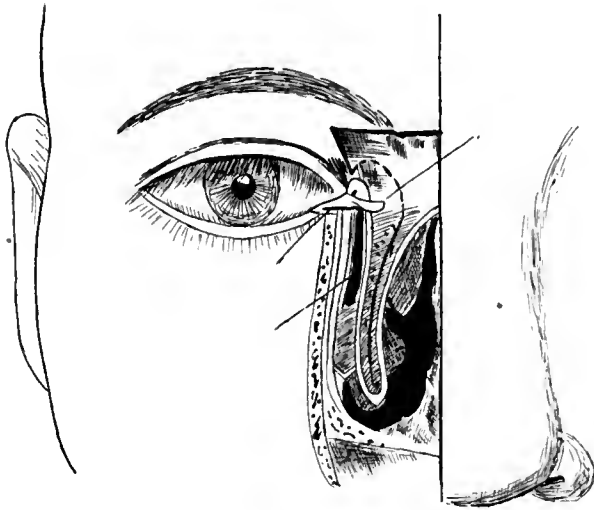


Figure A

presses the sac into the nasal chamber through the opening made in the bone. My observation has been that it is sometimes difficult to grasp the sac without also engaging the probe in the bite of the grasping forceps. To overcome this slight difficulty (and at times it has been very annoying), I have caused to be made a special punch forceps, of delicate structure, that may be used in the universal handle. My method is to push the sac into the nasal chamber, as shown in one of the accompanying drawings, and then make a slit in the sac. This slit is made well forward and the lower lip of the biting forceps is then pushed through this incision; it being an easy matter then to punch out the sac floor and thus make the resection desired.

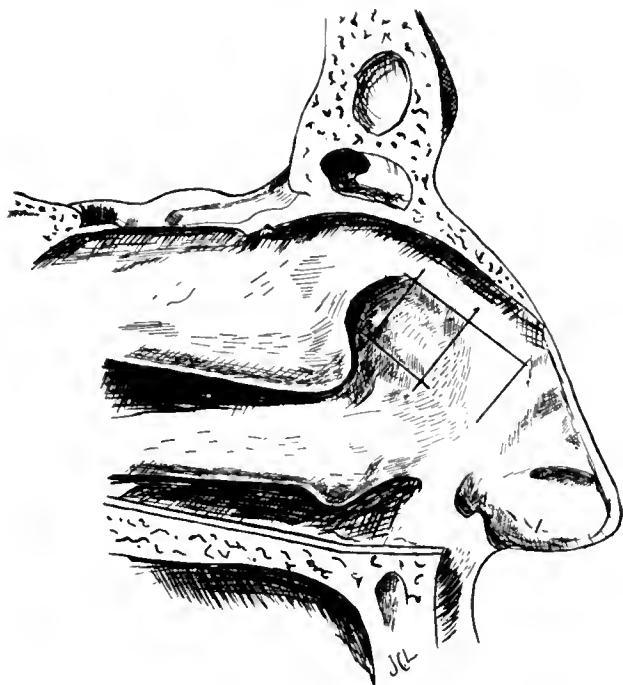


Figure B

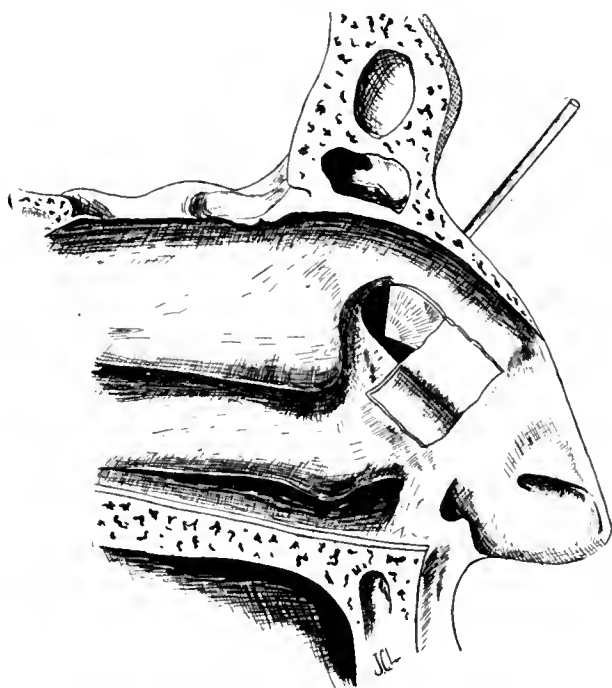


Figure C

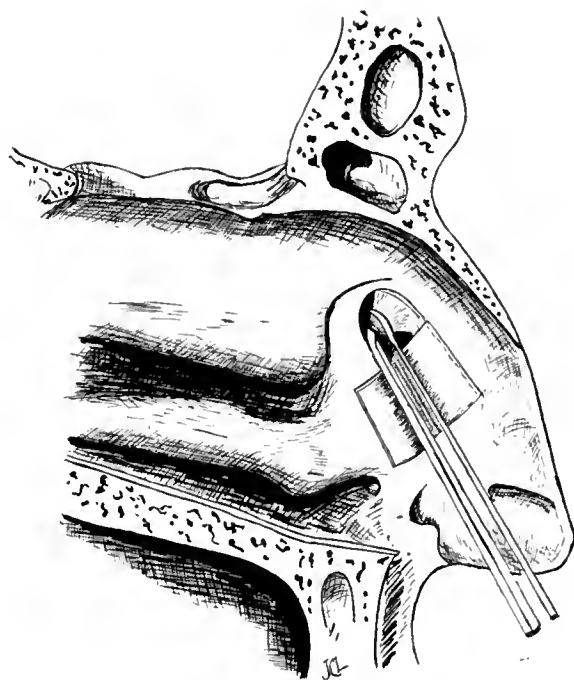
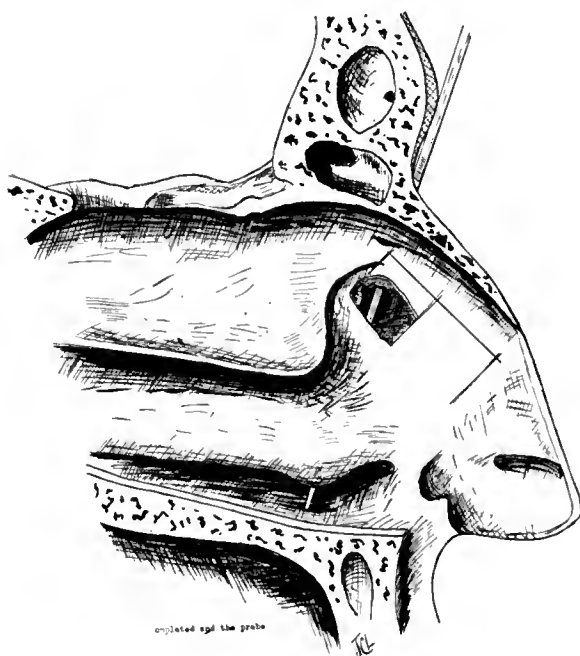


Figure D



enlarged and the probe

Figure E

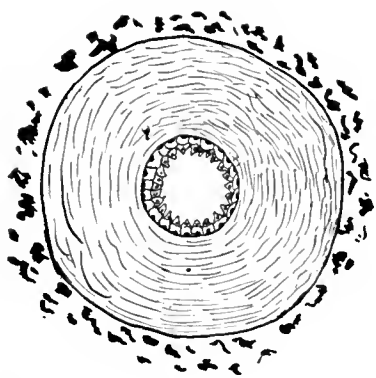


Figure F

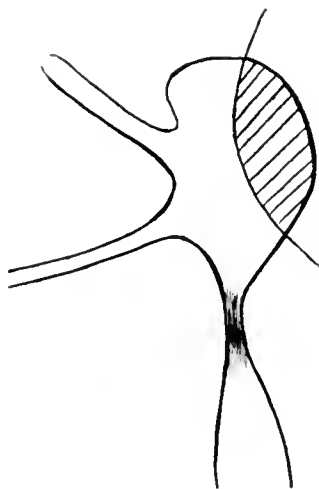


Figure G



Figure H

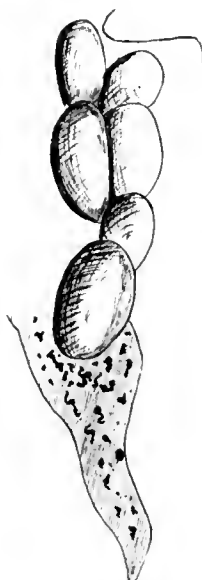


Figure I

FIGURES

Of the figures, Figure A shows the relation of the tear sac, canaliculus and duct to the surrounding tissues, cross sectioned. Figure B shows the relative positions and extent for the incisions. Figure C shows the flap of mucous membrane turned down so as to more readily admit light to the field of operation higher up in the nose. The probe is seen pressing the sac into the fenestrum made by the removal of the floor of the lacrimal fossa. Figure D shows the flap of mucous membrane replaced; the sac is grasped by the forceps and is being cut off by the knife. Figure E shows the operation completed and a probe inserted in the modified nasal duct. Figure F, in cross section shows the duct as it appears in the bony part of its course, high up under the middle turbinate. Figure G is a schema of the canaliculi with the sac, the shaded portion representing the part that is resected. Dark shaded portion just below the mouth of the sac, represents the usual point of stenosis. Figure H is a cross-section of the pyriform process, the shaded tip representing the portion that is sometimes sacrificed when it projects too far mesially. Figure I shows the usual relation of the ethmoid cells to the fossa lacrimalis.

DISCUSSION

DR. FRANK C. TODD, Minneapolis: Had the secretary asked me to open a discussion upon this paper instead of assigning the discussion without my knowledge, I should have declined, because I have never performed the operation of Dr. West, which Dr. Clark discusses, though I had the pleasure of hearing Dr. West describe his operation in London last year.

I agree with the writer that diseases of the lacrimal sac should be taken seriously and should receive early attention. There are some mild cases that can be cured by the old method of probing, but too few of the more severe cases are only temporarily relieved. Because of this fact, and because of the disagreeableness and long continuance of the treatment, it has been my custom for the past seventeen years in cases of stricture of the nasal duct to insert gold canulae carefully bent, cut and made to fit each case. These are usually left in place permanently and should be so placed that they are not visible. Styles I have found unsatisfactory. If styles are useful, canulae are better. In those cases which could not be relieved in this manner, I have been in the habit of extirpating the sac.

The operation described by Dr. Clark appeals to me very strongly. It should displace extirpation in nearly all cases. It is evident that it is an operation to be performed by the rhinologist. When I have seen Dr. Clark perform the operation I shall undertake it.

Dr. West deserves very great credit for having worked out the operation so carefully and persistently. It appears to mark a great advance in the treatment of these troublesome cases. We are indebted to Dr. Clark for his excellent presentation of the subject and I hope he will teach us by clinical demonstration how to perform the operation for I do not think it an operation to be undertaken without such instruction.

DR. J. C. BECK, Chicago: I had the pleasure of seeing Dr. Clark demonstrate his method, and I also had the privilege of seeing Dr. Yankauer's method, but the fact remains that the West operation as described is just like many of these delicate intranasal things. Where it sounds all right on paper, it is quite difficult to do, and if I had a suppurating tear sac I would want to be sure that the new opening was big enough and would remain permanently open. Therefore, I supplement the external operation with this proposition: a little bit of scar on the external surface will not make much difference. The technic that I follow is as follows: Expose the sac externally as usual, introduce a lacrimal probe, make a longitudinal incision into the sac, place a suture into each lip of this incision and employ them as retractors, incise the inner surface of the sac and expose the bony surface of the canal; by means of an electric burr make an opening into the nose, place a small rubber tubing through this opening and close the tear sac by means of the two sutures that were placed as retractors. The skin is sutured. The rubber tubing is allowed to remain for one week unless there is retention following. The subsequent treatment is to pass a large lacrimal probe through the slit lower canaliculus into the nasal cavity through the newly made channel.

DR. JOHN J. KYLE, Los Angeles: I want also to thank Dr. Clark for his interesting article and the beautiful illustrations. When Dr. West first described this operation it was impossible to get the instruments in this country, but I immediately undertook the operation by improvised instruments. I took the smallest Alexander gouge, a mallet and a small hook which we used for removing the ossicles, and with local anesthesia was able to do the operation without very much trouble. It was naturally very awkward and the tension great upon the operator, particularly when doing it for the first time. The patient was an elderly woman, and she suffered little or no pain. As to the result of the operation, about six months ago it was satisfactory with the exception that we had some cicatricial bands between the septum and the lateral wall of the nose. I have broken them down once or twice. I think where we have a very narrow nose it is best to do the submucous resection of the septum first, then put in strips of gauze and spread the cavity at the point of operation and avoid the possibility of any cicatricial bands. I have since secured the instruments and now I am ready to try it in a modern way.

DR. OLIVER TYDINGS, Chicago: This operation appeals to me as more simple than the Yankauer operation. I have done it on two cases recently, one of them a patient ninety years of age. I believe it is an operation that has a wide future. I did not understand from the description of Dr. Clark whether it was necessary to slit up the canaliculi. If that is so I wish him to mention it. I did not do it and don't think it necessary. Are any special instruments necessary? In my cases I did it with the Freer instruments. I hope he will bring that point out.

DR. EDWARD L. MEIERHOF, New York: I agree with Dr. Clark that a great many ophthalmologists are not able to do this operation because their experience does not justify them in doing it. For those who desire to supplement their treatment by the usual methods, that is, without removing the lacrimal sac, I devised an operation a few years ago by introducing a small curet into the lacrimal sac. This is a very small curet with small sharp teeth. The method that is required is simply to split the canaliculus its entire length and pass in a probe up to No. 12, 3 mm. Theobald. In cases of dacryocystitis the lacrimal canal is very easily dilated, much more so than when we have simple epiphora due to a narrowing of the canal. After this has been completed a conical dilator (a female urethral dilator) which comes up into a broad base from its point is passed into the lacrimal sac so that a wide opening is obtained. Skeel's 2 mm. serrated curet can

he passed into this widened canal, into the lacrimal sac and duct and curettage of the pyogenic surface of the sac and duct freely made in all directions. In addition to curetting this surface, an applicator with a screw end, on which absorbent cotton is wrapped tightly, is wetted with 2 per cent. nitrate of silver solution, and is passed into the curetted sac and canal and rubbed in. By this means I have been able to cure a number of cases in two or three weeks, when other usual means had failed. In spite of the wide dilatation of the canaliculus there was no epiphora. If necessary, the curettage or wiping out of the sac with swabs dipped in the silver nitrate solution may be repeated.

DR. CLARK (closing discussion): Dr. Tydings speaks of having heard me on this subject at the meeting of the Illinois Medical Society at its recent meeting held at Decatur, Ill., and that he did not recall that anything was said about special instruments and for that reason he had made his first operation after this method without the aid of the special instruments and asks if there are such. Special instruments have been devised by Dr. J. M. West. Originally there was a right and left grasping forceps for use in grasping the sac as it is protruded through the floor of the lacrimal fossa into the nose. I have changed this and made grasping forceps that can be used in a universal handle and this obviates the necessity for a right and left. One or two other features in regard to the instrumentarium have been made and these are exhibited by Mueller & Co.

With regard to the age, it is advised that this operation be not attempted on the very aged or infirm or those suffering from weak heart action. My experience has enabled me to operate upon a woman 75 years of age and I assisted in another in which the age was 7. Dr. Tydings says that his patient was 90 years of age.

Dr. Kyle spoke of bothersome synechiae that he encountered after his work on the sac from the nasal route. It is my opinion that West has covered this possibility in that he advises a submucous resection of the septum high up, where there is much tendency of the septum to hug the outer wall of the nasal space, and in the region of the torus lacrimalis.

Dr. Joseph Beck speaks of his work with the burr. He makes an incision on the outside and goes in with the burr. This of course is not an intranasal operation. He states that none of his cases presented a scar. It is my opinion however that one might have a scar after such an incision; and of course it is of not so much importance to the operator, but to the patient it is quite a factor, and a refined woman will say, "I cannot think of the possibility of a scar on my face." So in this regard and from a cosmetic standpoint the intranasal operation is quite an advance over the external method of extirpation. It occurs to me that the opening made on the outside, and where the drill is used, one might get some cicatricial changes that would later interfere with the proper function of the tear apparatus.

Regarding the obstruction at the valve of Hassner, it seems to me that if one had an obstruction at this point that a few probings would right matters.

Dr. Tydings did not understand that there was a previous slitting of the canaliculus. This is done preferably a day or two previous to the intranasal work. It is done in advance so that the edges of the canaliculus will not adhere and will readily admit the small probe. It is quite essential that the probe be used, for it greatly aids one in getting the sac into the window made in the floor of the lacrimal fossa, and the further orientation that is necessary.

West reported upon his work in the clinic of Professor Silex at Berlin at the meeting of the Congress of Surgeons held in London this year. He gives his percentage of cures as being 90.

SUBPERIOSTEAL BLOOD CYST (?) OF THE ORBIT SIMULATING OSTEOSARCOMA

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The rarity of such cases as this herein presented is, I believe, a sufficient warrant for its presentation.

Perusing such case reports as could be found which from their title suggested even a possibility of similarity to the case herein to be described, I found none like it, and the only one approaching it was reported by Denig in 1893. The report was very meager. It recorded a history of injury while fencing ten years previously, and some years afterward a tumor formed; when opened by accident the tumor extruded a brownish fluid. From the description one might say the tumor could not have been angioma, but very probably a blood cyst located under the periosteum of the frontal bone.

Among cases reported in French, German, Russian and Italian I could find only a few which might be mentioned because from the title one could suspect a similarity. Baquis reported a case in 1893, which is cited by Denig. A boy of 11 years had been injured only a short time before he presented himself for examination. There was concomitant fracture of the frontal bone. Gourlay's case was one of cavernous angioma. Brandés' case was one of spontaneous hemorrhage in scurvy. Bergman's case was one of oil cyst of the orbit. Ginzberg's had a serous cyst of the orbit containing red and white blood cells and cholesterin, but it was not subperiosteal. Impoff's case was a cavernous angioma beneath the external rectus. Bajardi had a case with no history of injury, in which the cyst contents was chocolate colored, there being megalocytes and yellowish cholesterin crystals, also hematoidin crystals.

Many other cases on investigation proved to be cavernous angiomata. The foregoing only show similarity in that the fluid contents had somewhat the same color and consistency as in my own case.

The following is a report of my case:

F. W., white man, aged 38, army employee, of Fort Washington, Md., came under my notice about Sept. 1, 1913, through

the kindness of Maj. Van Poole, then stationed at Fort Washington.

The previous history is as follows: Three and a half years ago he first noticed that the left eye was bulging and then consulted a physician in Florida, where he was stationed. The doctor said it was "watery tumor of the bone." Patient was given increasing doses of drops, probably potassium iodid. He was treated one year. The tumor got larger and began to pain before the year was out. He first felt it ten months after the swelling appeared.

He was ordered to Boston. There no treatment was advised. He remained three months.

He came to Washington, consulted an oculist, and was treated with medicine internally several months, but got no better.

Maj. Van Poole next undertook the case, administering medicine internally for six months, but as sight was rapidly failing he brought the patient to me in consultation, and operation was advised and accepted. The major's examination showed vision in the left eye was 20/100-2 and the field was much contracted. After operation the patient told me he had consulted a well-known oculist in Baltimore who said he had an inoperable tumor of the bone and refused to operate, but offered to give him internal medication.

The only other bit of history which seems to have some bearing on the case is that during the summer of 1909 he made a deep dive, possibly twenty-two feet, striking his head a glancing blow against some projecting iron object while going down. About six months later the aforesaid swelling began.

On Sept. 8, 1913, the patient came to my office and I found with the exophthalmometer intertemple 115 mm., O. D. 18, O. S. 27, which measured a very marked protrusion of the left eye. Vision O. D. 20/20 +; O. S. 20/100 —; peripheral vision very poor. On palpation the tumor was hard above and to the outside of the eye and just under the supra-orbital notch there was a point soft to the touch but not fluctuating, as if the lacrimal gland had been displaced forward and inward.

On Sept. 9 at 2 p. m., the following operation was performed, morphin $\frac{1}{4}$ gr., atrophin $\frac{1}{150}$ gr., having been given one hour before the general ether anesthesia was administered.

The initial incision began over the temporal ridge of the frontal bone about two inches above the zygomatic process of the malar and extended to this process, turning and paralleling the lower border to within one-half inch of the tragus.

The Krönlein operation was then begun and progressed to the cutting of the orbital plate. However, as this was being completed preparatory to cutting the zygoma, a small perforation was accidentally made in what subsequently proved to be the capsule of the tumor and there was extruded the most wonderful matter I had ever seen, entirely unlike anything known to me. The consistency was like that of honey, the

color was mahogany brown, thickly sprinkled with yellow crystals, dustlike in arrangement. There was no odor. The only substance which reason suggested was old blood containing cholesterol crystals colored by long contact with the hematin of the blood. Some of the matter and as much of the capsule as could be recovered intact was sent that afternoon to the microscopic laboratory of the Army.

The Krönlein operation was here abandoned, the left eyebrow painted with tincture of iodine and an incision made from the median end of the eyebrow, through its full length to intersect the first incision. The periosteum was now raised above and well up over the frontal bone, and downward toward the



Subperiosteal blood cyst of the orbit simulating osteosarcoma.

orbit. The color of the bone overlying the frontal sinus was bronze ivory and it was as thin as paper, breaking on slight pressure much as does the shell of a paper-shell almond.

The tumor was opened wide by an incision and more than half an ounce of matter similar to that first seen was obtained. The capsule, which was attached subperiosteally, was then removed by slow dissection. The measurements of the cavity from which it was extracted were found to be $1\frac{1}{4}$ by $1\frac{3}{4}$ by $2\frac{3}{4}$ inches.

The anterior plate of the frontal bone was removed from one-quarter of an inch to the nasal side of the supra-orbital notch to the external angle and the superior wall of the orbit, usually found forming the floor of the frontal sinus, was found completely eroded from upward pressure of the tumor.

Inspection of the cavity remaining after the removal of the tumor showed that it communicated directly with the upward expansion of the infundibulum of the frontal sinus, the lining membrane of which was normal glistening gray. The frontal sinus had evidently been separated from the tumor by only the capsule and there was no history of any discharge or other sign of inflammation of the sinus. Fluid easily passed through the infundibulum into the nose.

The wound was packed with iodoform gauze, closed with sutures and only a passage at the temple for a gauze drain was left unsutured. Within twenty-four hours there was delirium from the iodoform, which disappeared when the packing of the wound was removed and the wound irrigated with sodium chlorid and repacked with sterile gauze. Otherwise the patient made an uneventful recovery.

When last seen, Feb. 7, 1914, the vision in the left eye was 20/40 + and with a slight spherocylinder correction he was able to get 20/20 +. Exophthalmometer intertemple 115 mm.; O. D. 18; O. S. 21. The peripheral vision had likewise improved.

The specimen for microscopic examination was, as I have said, given to Maj. Van Poole and sent to the laboratory of the Army. But owing to the fact that it arrived during the absence of the officer in charge of the laboratory the contents of the cyst was never shown him and was lost, so that his report is not complete. However, from the cyst wall he deduced the probability of a melano-angiosarcoma. This is of course open to question, and when I described to him the appearance of the contents, he frankly stated he did not know what it could be.

I can see no good reason why the tumor may not have started as a hemorrhage, have become encysted and then later taken on an angiosarcomatous character, which would account for the increase in size and proptosis. Of course the melanotic character may easily be due to the long contact with contained blood pigment.

Among other facts of note are the complete capsule which was nourished by capillaries only and its location beneath the periosteum. There was no hemorrhage during its extraction. Yet the supra-orbital artery and vein bled when severed during the operation, thereby showing no connection with the tumor. Baquis' case gives a suggestion as to the origin of the tumor although there was no sure or certain evidence at the operation of my case of any previous fracture of the frontal bone.

In discussing the foregoing case it would seem proper to consider first, the possible cause; second, the fact of injury occurring some months prior to any noticeable swelling (except such as occurred at the time of injury and subsided during the next two or three weeks, i. e., normally); third, gradual enlargement of the tumor together with the increase of pain, the history of almost constant iodid medication without any relief;

fourth, the apparently excusable diagnosis of osteosarcoma and the refusal to operate in spite of repeated requests of the patient; and fifth, the successful cure following the operation, which was begun with the expectation of alleviating the patient's suffering at least for a time and the hope of a cure by radical excision of all apparently diseased tissue, as had been my good fortune in some cases of true osteosarcoma operated on at an earlier date.

In the first place should come this statement taken from Adami: "Extensive hemorrhages into the substance of sundry organs may result, not in the ultimate absorption of the exuded fluid but in cyst formation. The hemorrhage leads to the destruction of the tissues of the infiltrated area and eventually a capsule is formed around the exuded blood." Did this case of mine act like a blood cyst? No, it did not, for it did not grow smaller or remain stationary. It did, however, become enlarged and caused erosion of a large part of the frontal bone and discoloration of another part. Yet the tumor itself was encapsulated. Nevertheless the proptosis increased and the patient grew deformed and an object of annoying solicitation and critical comment.

Injury, it would seem, had been a disturbing element of the vessels and the surrounding connective tissue. The natural sequence of the increase in the growth of both these tissues was the enlarging and increasingly painful tumor.

The failure to operate earlier and so relieve the patient of his anxiety and the tumor simultaneously, is, I believe, excusable, as is also the prolonged iodid medication administered by several good men and excellent surgeons. Yet how much better can we looking backward now see, "*post hoc, ergo propter hoc*": for we realize what a comfort and freedom from desperate anxiety would have been the patient's portion brought about by an early operation. Furthermore, suppose the tumor had been an osteosarcoma, would not the chances of a permanent cure have been many times greater through early radical operation? Surely that has been our experience.

In conclusion let me take my place beside the conservationists (those who believe in early operation on obscure cases). Something can be learned from exploration and many cases could be cured. I am sorry not to be able to have the patient here and must therefore use his photograph as a substitute. I

may say furthermore, that he is hale and hearty and enjoys good vision in what very soon, undoubtedly, would have been a blind eye.

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AN ABSCESS OF THE OPTIC NERVE*

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Visual disturbances dependent on malignant growths in the accessory sinuses or the nose now form a well-recognized clinical picture and can be diagnosed without great difficulty. The blindness, either relative or absolute, may result from actual pressure of the orbital periosteum on the optic nerve. On the other hand, causes other than pure pressure may be the source of the trouble, as the case here described will show.

J. K., 55 years old, was seen in the surgical clinic in Graz in December, 1892, by Professor Elschnig, whom I wish to thank for his kindness in allowing me the use of this material. For over a year he had been suffering from a carcinoma of the right antrum of Highmore, which completely filled the nose. A few days before the ophthalmoscopic examination, he became blind suddenly. There was an exophthalmos of about 8 to 10 mm. The pupil was dilated and reacted only consensually and to convergence. Ophthalmoscopically, no pathological changes could be found. The left eye was normal. About one month later the patient died.

At necropsy, the right optic nerve at the base of the skull was found to be thickened as far back as the chiasm. The carcinoma, which in places was suppurating, had broken through into the orbit at the apex and was pressing on the nerve. At the bulbar end, the nerve sheaths were swollen into an ampule form, such as is usually seen accompanying choked disk due to intracranial pressure. The remaining intra-orbital portion of the nerve was normal. During the dissection the roof of the orbit was lifted away and the entire course of the nerve exposed. The intracanalicular portion of the nerve was swollen as much as its bony limitations would allow. Here the dura on the nasal side was involved in the tumor. On endeavoring to separate the nerve from the new growth, the dura burst and there was evacuated a small amount of creamy pus that contained Gram-positive diplococci.

Figure 1 is a diagrammatic sketch of the course of the optic nerve from the eyeball to behind the chiasm, the superimposed numbers giving the lengths of the various portions in millimeters. Sections through the nerve, designated by the capital letters, are described in the following paragraphs. The con-

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striction between D and E represents the intracanalicular portion of the nerve.

A. Histologically, the eye in its anterior aspects showed no pathological changes. The papilla presented a deep physiological excavation. A conus existed laterally, and on the opposite side of the disk was a supraposition of the retinal pigment with the lamina elastica of the choroid. The perforating scleral vessels were possibly slightly dilated, but there was no infiltration of their walls.

B. The structure of the lamina cribrosa was loose, as was that of the nerve fibers directly behind it. This looseness led to the formation of small empty spaces within the nerve-fiber bundles. These were identical with those described by Axenfeld and others in myopic eyes and claimed by them to be similar to the Schnabel "glaucomatous caverns." There is, however, but little similarity between the true glaucomatous and the myopic caverns. This portion of the nerve was poor in glia tissue, but the connective tissue trabeculae were thickened and

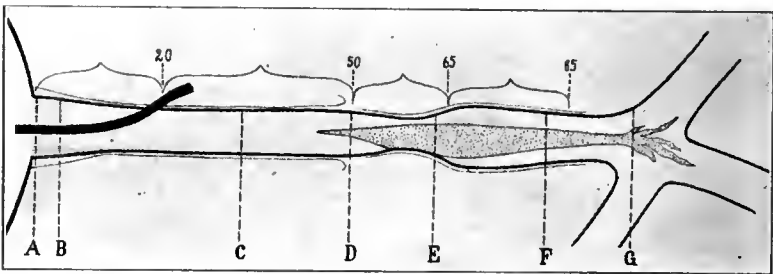


Fig. 1.—Schematic diagram of abscess of optic nerve.

nuclear-poor. The vessels were slightly thickened and perhaps somewhat fuller than usual, and the arteries showed a moderate degree of arteriosclerosis. The Pal-Weigert stain revealed a slight atrophy of the papillomacular bundle alone while the rest of the nerve-fiber bundles were normal. The picture was that of a simple descending atrophy of the optic nerve at its bulbar end.

C. Farther back toward the beginning of the abscess the picture changed somewhat. The dura was sensibly thickened, while the arachnoid and pia showed but few, if any, abnormalities. The connective tissue septa were markedly thickened, rich in nuclei and compressed the nerve-fiber bundles even to the point of obliteration. These bundles were smaller than normal and some of their medullary sheaths were entirely gone. Pal-Weigert stain showed some medullary degeneration products in the papillomacular bundle alone. The vessels were thickened and slightly dilated, but not infiltrated.

D. The front end of the abscess was located centrally in the nerve and was surrounded on all sides by nerve fiber bundles.

It presented a broken-down area that increased in size as the sections proceeded posteriorly. This area was filled almost exclusively with mononuclear leukocytes, although some polymorphonuclears were present. A great deal of fibrin and detritus was to be seen. The nerve-fiber bundles were pressed to all sides by the abscess and were distinctly smaller than normal. The medullary sheaths had partially disappeared and many broken-down products of these sheaths came to view with the Pal-Weigert stain. A few fat granules were to be seen in the periphery.

E. In the central region of the abscess, that is, at the junction of the canalicular and basal portions of the optic nerve, the nerve was markedly thickened. On the nasal side of the canalicular portion the dura had entirely disappeared and the arachnoid and pia were thickened and infiltrated. However, even with special stains, I was unable to detect any organisms in the sheaths or abscessed portions of the nerve. But it must be added that the specimen had been hardened for a long time in Mueller's fluid, which destroys the staining power of the bacteria. Here the abscess extended from the nasal side of the nerve nearly through to the temporal side with the exception of a small bundle of nerve fibers, crescentic in shape, adherent to the temporal sheaths. In the abscess proper, only leukocytes, fibrin, cell-detritus, and degenerated medullary sheaths could be found. The predominating type of leukocyte was the mononuclear. A few fat-granule cells became visible with the Pal-Weigert stain. The temporal nerve-fiber bundles, still present, were compressed by the thickened connective-tissue septa, but showed no distinct inflammatory changes. Separating this seminormal area from the abscess, was a broad band of fibrin, incompletely encircling the necrosis. This fibrin had also forced its way in between the nerve-fiber bundles in various localities. No carcinomatous cells were to be found.

F. More posteriorly the abscess became excentric. Practically the same picture was presented here as toward the front end of the abscess. The Pal-Weigert stain, however, showed more degenerated nerve fibers here and smaller bundles than anteriorly. The intima of the vessels seemed to be more sclerotic while their walls were thickened and somewhat infiltrated. Around some of the vessels were mononuclear leukocytes in such quantities as almost to form a sheath.

G. Horizontal sections through the chiasm showed the abscess to lie fairly in the center of the nerve. The abscessed area was here rather broad and not sharply circumscribed, in that numerous bypaths could be seen diverging from the main area. The neighboring nerve-fiber bundles were compressed and nuclear-poor, as well as fiber-poor. Mononuclear leukocytes were found around the abscess area, forcing their way in between the thickened connective-tissue trabeculae. As seen in the schematic diagram, the abscess was not confined to the involved nerve alone, but forced its way through the center of

the chiasm along the crossed bundles, so that it impinged on the opposite tract. The abscess came to a sharp point here and ended. Throughout the entire chiasmic area involved, the vessels were dilated and their intima was richer in endothelium. They were filled with red and white blood-cells, and their walls were to a great extent infiltrated.

We have, then, an abscess of the optic nerve, extending directly from a suppurating carcinoma that filled the nose. The dura was first attacked in the intracanalicular portion and drawn into the abscess, following which the abscess attacked the optic nerve proper. The pus burrowed anteriorly and posteriorly in a conical form. Anteriorly the globe was not reached, while

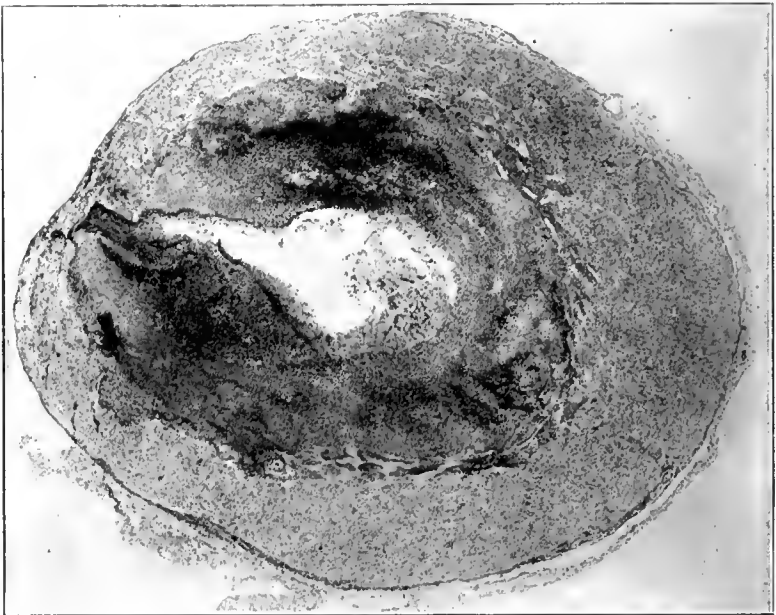


Fig. 2.—Cross-section through abscess of optic nerve.

posteriorly there seemed to be less resistance and the abscess involved the chiasm.

Apart from the rare clinical and still rarer histological findings, the case is of interest in that it throws light on the scotomata and blindness, secondary to accessory-sinus disease. But a glance at the normal anatomy is necessary first. After the central artery and vein leave the nerve, about 20 mm. posterior to the globe, there is a short stretch of nerve without any large central vessels. In this area and posterior to it, small veins run from the surrounding sheaths and nerve-fiber bundles toward

the center of the nerve, finally uniting to form a small central vein, the vena nervi optici centralis posterior. Toward the basal end of the canalicular portion of the nerve, this vein assumes a fair size and is fed by venules from the nerve proper, as well as from the nerve sheaths. It finally empties directly into the cavernous sinus. The first anatomical description of this vein was published by Kuhnt, and it was later mentioned in a pathological connection by Vossius.

In the bony optic canal, the ophthalmic artery lies below the nerve and is embedded in the dura, which fulfils the double function of optic nerve sheath and periosteum. The true periosteum lining the orbit, blends with the dura at the orbital end of the canal and loses its identity. Hence any infection of the periosteum can pass directly into the dura and thence, through the posterior central vein, into the center of the nerve itself.

Due to this vascular arrangement, the canalicular portion of the optic nerve is peculiarly susceptible to inflammations originating in the dura, either intracanalicular or intra-orbital. As the "Kuhnt vein" lies in close proximity to the papillomacular bundle, any inflammatory involvement of the vein would lead primarily to a central scotoma and eventually to blindness. Any infiltration around the vein, with a subsequent destruction of the papillomacular bundle within the canalicular portion of the nerve, would result in a descending degeneration of this bundle. Such a case was reported by Elschmig in his work on "Stauungspapilla."

An important observation in this respect was made by Elschmig in a case of chronic internal hydrocephalus with choked disk. There existed a diffuse meningitis, which included a chronic inflammation of the three sheaths, especially in the intracanalicular portion. Only around the posterior central vein and its accompanying small arteries was a massive lymphocytic infiltration, pressing on the papillomacular bundle. O. Sachs of Innsbruck made a similar observation in a case of intoxication neuritis. The appearance of a central scotoma as an early symptom is thus explained by the histological findings.

Based on this anatomy and on the reported anatomical findings, we are justified in assuming that the majority of visual troubles, secondary to accessory sinus disease, result from a direct attack on the canalicular portion of the optic nerve or

its sheaths. This may be a simple edema, transient in effect; an infection passing from the periosteum through the sheaths into the nerve; or a direct necrosis, such as was found in this case.

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ECONOMICS OF THE EYE, EAR, NOSE AND THROAT

TOGETHER WITH THE ECONOMICS OF THE ENTIRE BODY
(PHYSICAL ECONOMICS)

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¶ 1. A scientific method for the determination of the economic loss from damages to the eye, ear, nose and throat must provide for the measurement of each of these separately, or together, and also in conjunction with the measurement of the economic loss from damages to any other part of the body which was caused from the same injury. A method that could be used for only one set of organs, like the eyes, and could not be used to determine the economic loss from damages to the functions of other parts of the body which were caused by the same injury, would not be a scientific method, because the damages to the other parts of the body would have to be estimated; and, therefore, the method would be partly scientific and partly empirical, making it, as a whole, empirical, since it would be like a chain which is no stronger than its weakest link. A scientific method must also include one for the determination of the economic value of man from his earning ability. This necessitates the analysis of the earning ability according to the natural-science method, in order to determine its indispensable elements, which may be used as factors in a mathematical formula for the purpose of ascertaining its true value, which will agree with all the existing conditions of these factors. This analysis of the earning ability shows that its first indispensable element is the function of the system and organs of the body, namely, the functional ability of the body. The functional ability of the body would be of no value in the earning ability unless the mind and body were trained to perform some kind of work successfully. This training of the mind and body to do work successfully may be termed the

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technical ability. It is acquired largely during the growth of the body and is dependent on its functional ability for its fullest development, and therefore it should be taken into consideration in ranking a pupil at school.¹ The third and last indispensable element of the earning ability of man, namely, the competing ability, or the ability which he has, after being possessed with a good functional ability and after having acquired a good technical ability, to secure a position in life or establish one for himself and then to perform its duties successfully enough to obtain an income therefrom sufficient to warrant the whole expense of his education with at least the ordinary profits of an equally valuable capital.

¶ 2. Therefore, this analysis of the earning ability discloses its three indispensable elements as follows: (1) The functional ability; (2) the technical ability; (3) the competing ability. These indispensable elements of the earning ability, according to the natural-science method, may be used as factors in a mathematical formula for the purpose of getting at the value of the earning ability in its relation to the actual existing conditions of these indispensable elements. As the three words, expressing each of these three elements, would be too bulky to use in a mathematical formula, we avoid it by letting each be represented by a symbol as follows: (1) The functional ability to be represented by F ; (2) the technical ability to be represented by T ; (3) the competing ability to be represented by C ; and (4) the earning ability to be represented by E . Expressing these factors as actually multiplied, by using the sign of multiplication, we have as follows: (1) $F \times T \times C = E$, the mathematical formula for the normal earning ability of the body and for determining the economic loss from damages to the body from injury or disease.

¶ 3. In order to get at the value of E , the earning ability, according to the actual existing conditions of its indispensable

1. We might mention that this is being done at the University of Maine, where the functional ability of the student and the technical ability of the student are considered as factors of the efficiency of the student, that is, the functional ability, F , multiplied by the technical ability, T , equals Ef , the efficiency of the student. For instance, if a student had, according to the test given, a functional ability of say 0.80, and a ranking according to the test given, of say 0.75, for his technical ability, the formula would stand as follows: $0.80F \times 0.75 T = Ef$. Hence the efficiency of the student would be 0.60. If at the end of the four years' course the factors stood, according to similar tests, as follows: $0.90F \times 0.90T = Ef$, the efficiency would be 0.81, that is, he has made a gain, during the four years' course, of 0.21, on a base of 0.60, or 35 per cent. gain. By this method the gain in efficiency between the beginning and the end of the four years ending in 1908, and every year since that date showed that it was those students who made the greatest effort to improve their physical and mental conditions that had the greatest percentage of gain, and therefore won the prizes.

elements used as factors in this formula for the normal earning ability of the body, it is necessary to analyze the first indispensable element, namely F , the functional ability of the body, according to the natural-science method, by resolving it into its component parts and thereby determine its indispensable elements, which may be used as factors in a similar mathematical formula to obtain its value according to the actual existing conditions of these indispensable elements. In this analysis the functional ability of the body is resolved into its component parts, in the same manner as the earning ability was resolved into its component parts, namely, by selecting elements which are so interdependent that each is needed to insure the function of the other, and these, taken together as factors and multiplied, are just as essential to obtain its true value as are the prime factors of a composite number to be multiplied together to obtain that number. Indeed the process is identical, and for this reason the analysis is absolutely correct, because by computing the value of the body by using these indispensable elements as factors, we obtain a result which is correct mathematically, and which agrees exactly with all the existing conditions of these indispensable elements of the functional ability of the body. By this analysis, systems and organs have been selected and grouped together into four units (a, b, d, g) in accordance with their development and associated functions. We thus have four units, which include the functions of the structures of all the indispensable elements of the body, which may be used as factors in a mathematical formula to determine the value of each unit, and therefore the whole functional ability of the body, according to the actual existing condition of the indispensable elements of those units.

Again we analyze each of these four units, (a, b, d, g) by resolving each unit into its component parts in the same manner as the earning ability and the functional ability of the body were resolved into their component parts, namely, by selecting elements which are so interdependent that each is needed to insure the functions of the other, and these, taken together as factors and multiplied, are just as essential to determine the true value of the unit as are the prime factors of a composite number to be multiplied together to obtain that number. We thus have resolved each of the four units into their component parts, ($a = hik$; $b = mnp$; $d = qrs$; and $g = uvw$), which is detailed sufficiently for all computations in this prob-

lem, making twelve divisions, each of which includes all the indispensable elements of the units and therefore of the functional ability of the body. Each of the three divisions of a unit must be used as a factor in order to determine its true value, according to the existing conditions of the indispensable elements of these divisions, the same as the prime factors of a composite number are multiplied together to obtain that number, which is the same as the four units which are used as factors and multiplied together to obtain the functional ability of the body; and finally which is the same process as when the functional ability, the technical ability, and the competing ability are used as factors and multiplied together to obtain the earning ability of a person, according to the existing conditions of these indispensable elements. Thus, all these values in this problem are obtained by one uniform method, which corresponds precisely to that which is employed in the natural sciences to obtain the value of any physical force.

¶ 4. Again, as the words expressing the indispensable elements of each unit and their subdivisions as factors would be too bulky to use in a mathematical formula, we let each be represented by a letter as a symbol. No letter of the alphabet is used as a symbol that would lead to confusion in the solution of this problem. They are as follows:

$$F = \begin{cases} a = \left\{ \begin{array}{l} \text{Osseous, articular and} \\ \text{muscular systems,} \\ \text{consisting of} \end{array} \right. \begin{cases} h, \text{ the bones} \\ i, \text{ the ligaments} \\ k, \text{ the muscles} \end{cases} \\ b = \left\{ \begin{array}{l} \text{Circulatory and respira-} \\ \text{tory systems, consist-} \\ \text{ing of} \end{array} \right. \begin{cases} m, \text{ the vascular system} \\ n, \text{ the blood} \\ p, \text{ the lungs and their accessory organs} \end{cases} \\ d = \left\{ \begin{array}{l} \text{Digestive and Genito-} \\ \text{urinary systems, con-} \\ \text{sisting of} \end{array} \right. \begin{cases} q, \text{ the alimentary canal and its accessory organs} \\ r, \text{ the kidneys with the genital organs} \\ s, \text{ the skin} \end{cases} \\ g = \left\{ \begin{array}{l} \text{Cerebr. system, nerves} \\ \text{and organs of special} \\ \text{sense, consisting of..} \end{array} \right. \begin{cases} u, \text{ the brain, its membranes and its nerves} \\ v, \text{ the spinal cord, its membrane and its nerves} \\ w, \text{ nerves and organs of special sense} \end{cases} \end{cases}$$

This analysis is complete because each division of a unit as a factor of the unit, the same as each unit as a factor of the functional ability of the body, satisfies the requirements of our definition. Each factor is composed of the functions of systems, or organs, or both, which are so interdependent that each is needed to insure the functions of the other. The factors of a unit taken together and multiplied determine a true value for the unit, in the same manner as the units as factors taken together and multiplied determine a true value for the functional ability of the body, and finally the functional ability, with the technical ability and the competing ability of the body as factors,

determine a true value for the earning ability. With the earning ability obtained according to the existing condition of its indispensable factors, we are able, by the law of average, to determine the economic value of man just as accurately as those values which are the bases of life insurance, one of the largest businesses of the world. This is sufficient and accurate enough to meet and satisfy every condition. Furthermore, if we have a method sufficient and accurate enough to obtain a true economic value of man, as a whole, we certainly have one sufficient and accurate enough to obtain a true value for any fractional part of the whole, for the whole includes all of its parts.

¶ 5. To recapitulate, we include all the functions of the structures of the systems and organs of the body in the four units, each of which is absolutely indispensable to F , the functional ability of the body, and by the symbols representing them as factors we are able thus to form the following: (2) $a \times b \times d \times g = F$, the mathematical formula for determining the functional ability of the body according to the actual existing conditions of the functions of the structures of these units. By this analysis we are also able to determine the value of each of these units of the body in a similar manner as follows:

$$(3) h \times i \times k = a$$

$$(4) m \times n \times p = b$$

$$(5) q \times r \times s = d$$

$$(6) u \times v \times w = g$$

These are the mathematical formulas for determining the functional ability of the units according to the actual existing conditions of the indispensable factors of these units.

¶ 6. In normal conditions of health the coefficient of F , the functional ability of the body, is equal to one. It follows, then, logically, that for that particular person the coefficient of T , the technical ability, and of C , the competing ability, is also equal to one. Hence E , the earning ability, would be normal and depend on the income derived from the vocation followed. From this income, the age of the person, and the rate per cent. per annum used in the computations of the compound interest discount, the present economic value of that person may be obtained, as per Table 9, economic value of man. (¶ 63.)

¶ 7. When the coefficient of F , the functional ability, is amplified according to its analysis (¶ 4), we have the four

units, a , b , d , and g as its coefficient, thus: $(abd g) F$. As the coefficient of C , the competing ability of the person, is composed of the same elements as F , the functional ability of the body, it must have the same coefficient, thus: $(abd g) C$. We would thus have for the amplified formula as follows: (7) $(a \times b \times d \times g) F \times T \times (a \times b \times d \times g) C = E$. In case of damage to the body from injury or disease, the condition of the coefficient of unit a , as one of the factors of the coefficients of F , would be determined by the formula, (3) $h \times i \times k = a$; the condition of the unit b by the formula, (4) $m \times n \times p = b$; the condition of the unit d by the formula, (5) $q \times r \times s = d$; and finally the condition of the unit g by the formula, (6) $u \times v \times w = g$.

¶ 8. In using the mathematical formula for the normal earning ability of the body for the purpose of determining the economic loss from injury or disease, T , the technical ability, may be omitted, because this factor of the earning ability is not injured but limited in proportion to the damage to F , the functional ability of the body, whose impairment includes these damages. The coefficient of T , the technical ability, after it becomes fixed in life, remains 1, or normal, for that particular person, and for this reason also may be discarded, because multiplying any number by 1 does not change its value. We then have the mathematical formula for the normal earning ability of the body reduced to two factors as follows: (8) $F \times C = E$, the mathematical formula for determining the economic loss from damages to the body from injury or disease. The formula in this form corresponds to the simple terms of arithmetic; namely, F , the functional ability of the body, corresponds to the multiplicand, and C , the competing ability of a person, corresponds to the multiplier, while E , the earning ability of a person, corresponds to the product.

¶ 9. In formula (8) the coefficient of each of the factors F and C , and hence their product E , is equal to 1, and the exponents of F and C are also 1. As long as these values remain 1, it is not necessary to express them. When, however, a person meets with an accident and sustains damage to his body, the coefficient of F , the functional ability, and of C , the competing ability, immediately becomes less than 1. We then have to determine the status of F , the functional ability, by measuring its loss by scientific and economic standards of measurement, and thereby obtain its remaining functional ability for

its coefficient. This coefficient of F , the functional ability, becomes primarily the coefficient of C , the competing ability, according to ¶ 7. We then have to determine how this loss to F , the functional ability of the body, damages C , the competing ability of the person, in the vocation he follows, or any vocation he may be able to follow. If in a given case of damage to F the functional ability, for instance, like that of the loss of the left hand at the wrist joint, we determine that C , the competing ability, is damaged as much as F , the functional ability, then the remaining earning ability would be the product of these two factors as follows: The loss of the left hand at the wrist joint, according to Table 5 is 0.20. Hence subtracting this from 1, we have 0.80 for the coefficient of F , ($0.80 F$), the functional ability, according to ¶ 5, formula (2), which also according to ¶ 7, formula (7) becomes the coefficient of C , ($0.80 C$), the competing ability, and we then have according to ¶ 8, formula (8) as follows: $0.80 F \times 0.80 C = E$. Hence E , the earning ability, is 0.64 (Table 1, column 4), and the loss is 0.36, or 36 per cent. If it had been the right hand instead of the left hand, the loss to F , the functional ability, would be the same, but the loss to C , the competing ability, would be more, entailing a greater loss to the earning ability, and the person's economic value. Hence, the loss to C , the competing ability, would be greater than the loss to F , the functional ability, and we must therefore diminish the coefficient (the multiplier) of C , the competing ability, in order to diminish the earning ability (the product), to have it correspond with the actual conditions of the person. In order to diminish the coefficient of C , the competing ability, according to its own composite nature, namely, that of a composite number (as having been produced by its own indispensable factors to which nothing can be added nor subtracted without vitiating the principles of its formation and nature), we must multiply it by itself and indicate this process by a whole number, namely 2, for its exponent as follows: $0.80 F \times 0.80^2 = E$. Hence E , the earning ability, would be in this case equal to 0.512 (Table 2, column 2), and the loss would be 0.488 or 48.8 per cent. For these two conditions of C , the competing ability, just described, that is, when it is damaged to a severe or a nearly total degree, and the exponent will be 1, or a number greater than 1, we will let it be represented by a single (x) to indicate that it is to be determined in each individual case as follows: (9) $F \times C^x = E$.

¶ 10. If in any given accident, the damage to C , the competing ability, is less than F , the functional ability, in the vocation the person follows, then the coefficient of the former, the multiplier, must be increased to correspond with his ability to compete and earn more, and therefore, give a greater earning ability (product). If, for instance, in an accident there was produced a complete ankylosis of the left wrist joint, we find according to Table 5, that the loss to unit a is 0.10, and, therefore, there is 0.90 remaining functional ability, which, according to ¶ 5, formula (2), becomes the coefficient of F ($0.90 F$). This according to ¶ 7, formula (7), becomes the coefficient of C ($0.90 C$), the competing ability, and we have, according to ¶ 8, formula (8), as follows: $0.90 F \times 90 C = E$. As ankylosis of the left wrist joint would not prevent a person from following many vocations fairly successfully, C , the competing ability, would not be damaged as much as F , the functional ability, but to a slight degree. Therefore its coefficient (the multiplier) must be increased in order to have the earning ability (the product) increased to correspond with the actual conditions of the person. In order to increase the coefficient of C , the competing ability, according to its own composite nature, namely, that of a composite number (as having been produced by its own indispensable factors, to which nothing can be added nor subtracted without vitiating the principles of its formation and nature), we must find a factor of this fraction of the coefficient of C , the competing ability, which when multiplied by itself will produce it, namely, 0.90. To satisfy the conditions of C , the competing ability, from the damage to F , the functional ability, from ankylosis of the wrist, we determine that it is damaged to the first degree slight damage, which involves the finding of a factor of its coefficient which multiplied by itself ten times would produce it, and we indicate the process by a fraction, namely, $\frac{1}{10}$ for its exponent, as follows: $0.90 F \times 0.90 C^{\frac{1}{10}} = E$. Hence E , the earning ability, would be 0.8901 (Table 1, column 10), and the loss would be 0.1099 or 10.99 per cent. For this condition of C , the competing ability, that is, when it is damaged to a less degree than F , the functional ability, or to any one of the five degrees of slight damage to C , the competing ability, necessitating the finding of a factor of the fraction, which is always larger than the fraction itself, we indicate this process by a fractional exponent, that is by 1 divided by (x) to indicate

that it is to be determined in each individual case as follows: (10)
 $F \times C^{1/x} = E$.

¶ 11. We thus have two forms of the mathematical formula for determining the economic loss from damages to the body from injury or disease, which are also expressed in tabular form in Table 1 and Table 2, columns to the right numbered 10, 9, 8, 7, and 6; the computations are to be used when C , the competing ability, is damaged to a slight degree; columns numbered 5 and 4 of Table 1 and columns numbered 2, 3, 4, and 5 of Table 2 are to be used when C , the competing ability, is damaged to a severe degree, and columns numbered 6, 7, 8, 9, and 10 of this table are to be used when it is damaged to nearly a total degree, and when it is damaged to a total degree, 0 is to be used for an exponent; thus making this factor 0, therefore E , the earning ability, is 0, and the economic value of the person is 0 also.

¶ 12. In our analysis of the earning ability of man we have shown conclusively that the three indispensable elements, namely, (1) the functional ability, (2) the technical ability, and (3) the competing ability, must be used as factors and multiplied in order to obtain an earning ability which will agree with all existing conditions of its elements. In order to make this still clearer we will apply this analysis to one of the most common of physical forces, namely, a water-power, to show why an estimation of the loss to the functions of the body will not give the true loss to the earning ability, and therefore, why the economic loss from damages to the earning ability cannot be determined from the loss to the functions of the body alone. In the application of this analysis to a water-power, we will let the volume of water in pounds be the functional ability of water-power, because it is what nature gives to it, just as the body is what nature gives to man. The dam, the water-wheel and its connections to the countershaft, we will designate the technical ability of the water-power, because it is what man gives to it in consequence of his own technical ability, just as the technical ability is given to man by man. The competing ability of a water-power depends on its location and its ability to perform work in competition with other powers such as steam, gasoline, animal and man power. It will be seen that in order for a water-power to have an earning ability, it must have all three of these elements. There can be no question about the need of the first two elements, namely, the volume of

water in pounds going over the dam, and second, the dam, water-wheel, and its connections to a countershaft in order to give power. To illustrate the importance of the third, we will mention the fact that in Labrador there are some magnificent water-powers. These water-powers could be converted into an active, efficient force, by applying the technical ability of man in building dams and installing water-wheels and countershafts, thus delivering thousands of horse-power to be applied to machinery. These thousands of horse-power, however, would have no earning ability unless this machinery could be utilized in the manufacture of something useful for man in competition with other machinery run by water-power, or some other power, in other parts of the world. As there are so many other water-powers nearer the center of civilization which could be developed with less expense, and when developed would have a much greater competing ability, it is plainly evident that at present, the water-powers in Labrador, if developed, would have no competing ability, and therefore would have no earning ability, and hence, no economic value. This comparison of the earning ability of man with that of the water-power is so pertinent that it is plainly evident that the true earning ability of a person cannot be determined with any degree of accuracy or satisfaction, by a consideration of either his functional ability or his technical ability alone, or any estimation of them. We must have that other important element, the competing ability, used as a factor, in order to realize the earning ability in man the same as in a water-power. From this consideration of the subject, it is self-evident that neither the earning ability of man nor that of a water-power can be obtained from what nature gives to them alone, for without taking into consideration the technical ability which is necessary to develop them and the competing ability to realize their efficiency, there would be no earning ability, and hence no economic value.

¶ 13. Table 1 and Table 2 have been formed on the basis herewith set forth, of the composite nature of E , the earning ability of the body, and for the purpose of shortening and simplifying the computations in the solution of these problems. They have been found of such inestimable service in this respect that many have informed the writer that they considered the conception which led to their formation a great inspiration.

We have shown that we must not only ascertain the indispensable elements of E , the earning ability of the body, and

use them as factors in order to obtain a value which will agree with the actual existing conditions of these factors, but we must go a step farther and analyze its most important factor, namely, F , the functional ability of the body, in a similar manner and ascertain its indispensable elements and use them as factors to obtain a value for it which will also agree with the actual existing conditions of these factors. It will be seen, then, that the key to the right solution of this whole problem is the recognition of the composite nature of E , the earning ability of the body, and the composite nature of its principal factor F , the functional ability of the body. The key to the understanding of Table 1 and Table 2 and their right application in solving this problem is a knowledge of the exact condition of F , the functional ability of the body, as obtained by physical and laboratory examinations, standards of measurements and other data. When by these exhaustive examinations we find the indispensable function of the structures of each of the factors of a unit of the body are normal, or equal to 1, the coefficient of that unit is normal and equal to 1, ¶ 5, formulas (3), (4), (5) and 6(), and when each of the coefficients of the units of the body is normal, the coefficient of F , the functional ability of the body, is normal and is equal to one (¶ 5, formula 2). When, however, any of the indispensable functions of the structures of the factors of a unit become damaged from injury or disease, that factor becomes less than 1, its remaining value depending on the amount of damage its function has sustained.

We have arranged Table 1 and Table 2 in the form of a key to the solution of these problems, in which it will be seen that the computations for all degrees of damage to C , the competing ability, are comprised under four headings, namely, slight, severe, nearly total, and total. When the damage to F , the functional ability, causes only a slight or even a severe damage to C , the competing ability, the person may have an earning ability in the vocation followed equal to or greater than his personal expenses, and if so, he has an economic value. When, however, the damage to F , the functional ability of the body, is very severe or nearly total the damage to C , the competing ability, is so great that the person's earning ability is less than his personal expenses, hence such a person has no economic value. A person must have a remaining earning ability sufficient to pay for his own personal expenses before he has

any economic value, and the computations for very severe or nearly total damage to *C*, the competing ability, do not show it.

Table 1 gives in column 1 the loss to *F*, the functional ability of the body, from 0.01 to 0.80. When the loss to *F*, the functional ability, is from 0.01 to 0.80, the remaining functional ability is from 0.99 to 0.20, which constitutes the coefficient of *F*, the functional ability, and column 2. As the coefficient of *F*, the functional ability, primarily becomes the coefficient of *C*, the competing ability (§ 7, formula 7), its coefficient constitutes column 3, it being the same as column 2. When *C*, the competing ability, is damaged to the same degree as *F*, the functional ability, in the vocation the person follows, *E*, the earning ability, is the product of these coefficients, and this constitutes column 4. From *C*, the competing ability, being damaged to the same degree as *F*, the functional ability (the earning ability for which is given in column 4), and from its being damaged to the slightest or tenth degree less than *F*, the functional ability (as given in column 10), we divide into a scale of ten degrees for the purpose of grading the damage to *C*, the competing ability, when it is damaged less than *F*, the functional ability, in the particular vocation the person follows. The computations for *E*, the earning ability, for this purpose are given in columns 4, 5, 6, 7, 8, 9 and 10. The computations for the evolution of $\frac{1}{6}$, $\frac{1}{8}$ and $\frac{1}{9}$ were so nearly like those contiguous to them that they were omitted. The damage to *C*, the competing ability, in the two tables is, for convenience, divided into slight, severe and nearly total to total damage. The computations in the five columns from the right of this scale of damage in Table 1, namely, those numbered 10, 9, 8, 7 and 6 are to be used when the damage to *C*, the competing ability, is slight. The remaining two columns, namely, those numbered 5 and 4 are to be used when the damage to *C*, the competing ability, is severe. It will thus be seen that when any damage to *F*, the functional ability, causes a less damage to *C*, the competing ability of the person in the particular vocation he follows, and its coefficient (the multiplier) needs to be increased in order to increase *E*, the earning ability (the product), the computations necessary for this purpose are given in the scale of damage to *C*, the competing ability, the tenth degree of damage, carrying its value to as near 1 (when if it becomes 1 there would be no damage) as would ever be needed for practical purposes, because in every loss to *F*, the functional

ability of the body, as a rule, there would be some slight damage to C , the competing ability of that person.

From C , the competing ability, being damaged to the same degree as F , the functional ability (the earning ability for which is given in column 4, Table 1), and from its being damaged to a nearly total degree (as given in column 10, Table 2), we also divide into a scale of ten degrees for the purpose of grading the damage to C , the competing ability, when it is damaged more than F , the functional ability, in the particular vocation the person follows. The computations for E , the earning ability, for this purpose are given in columns 2, 3, 4, 5, 6, 7, 8, 9 and 10, Table 2. The computations in columns 2, 3, 4 and 5 in Table 2 of this scale of damage are to be used when the damage to C , the competing ability, is severe and 6, 7, 8, 9 and 10 when it is a nearly total damage. It will thus be seen that for all practical purposes Table 1 and Table 2 provide the complete computations in tabular form for all the existing conditions of C , the competing ability of a person, after loss to F , the functional ability of the body, from injury or disease, in accordance with the natural-science method, which is the only method that gives results which correspond with all the existing conditions of the indispensable elements of the body.

¶ 14. In connection with the reduction of the formula for the normal earning ability of the body, that is, in discarding T , the technical ability, as per ¶ 8, in formula (8), namely, $F \times C = E$, the formula for determining the economic loss from damages to the body from injury or disease, we alluded to the fact that when the value of the remaining functional ability and competing ability of the body was represented in the coefficient of these two factors, we had reduced the problem to the simplest terms of arithmetic, the coefficient of F , the functional ability of the body, corresponding to the multiplicand, and the coefficient of C , the competing ability, corresponding to the multiplier, and the coefficient of E , the earning ability of the body, corresponding to the product. The axiom in arithmetic is that increasing the multiplier increases the product and diminishing the multiplier, diminishes the product; that when the divisor is less than 1 the quotient will be greater than the dividend; and that the reciprocal of a number is 1 divided by that number.

In Table 1 the coefficient of C , the competing ability, has been increased (by evolution) in order to have E , the earning

ability, increased to correspond to the person's ability to compete more successfully in the particular vocation he follows than would be indicated by its coefficient, which is the same as that of F , the functional ability, as per ¶ 7, formula (7); and as C , the competing ability, is not damaged as much as F , the functional ability, its coefficient needs to be increased (by evolution ¶ 10), for increasing (the multiplier) the coefficient of C , the competing ability, increases (the product) E , the earning ability.

In Table 2, on the other hand, the coefficient of C , the competing ability (the multiplier), has been diminished (by involution ¶ 9) in order to have E , the earning ability of the body (the product), decreased whenever it is determined in a given case of injury that C , the competing ability, has been damaged to a greater degree than F , the functional ability of the person.

Table 1 represents the reverse of Table 2, and may be readily understood by the following illustrations. If we produce involution of the proper fraction $\frac{1}{2}$, or the decimal 0.5, to the sixth power, we express it as follows:

$$\begin{aligned}\frac{1}{2}^6 &= \frac{1}{2} \times \frac{1}{2} \times \frac{1}{2} \times \frac{1}{2} \times \frac{1}{2} \times \frac{1}{2} = \frac{1}{64} \\ 0.5^6 &= 0.5 \times 0.5 \times 0.5 \times 0.5 \times 0.5 \times 0.5 = 0.015625\end{aligned}$$

In this example one-half is the multiplicand and one-half is the multiplier. The product is just such a part of the multiplicand as the multiplier is a fractional part of 1, namely, it is one-half of it every time it is multiplied. This is the reason why raising a fraction by involution to any given power diminishes its value. This is what is done in Table 2, to the Coefficient of C , the competing ability, in order to have E , the earning ability, diminished to correspond to the condition of greater damage to C , the competing ability, than to F , the functional ability.

In Table 1 the reverse of involution is practiced, namely, evolution, and it is indicated by the reciprocal of the number, or one divided by the number, for the index of the exponent, or the root to be sought, as follows: $\frac{1}{64}^{\frac{1}{6}} = \frac{1}{2}$; $0.015625^{\frac{1}{6}} = 0.5$. If we did not wish to increase the value of the fraction so much, we would practice evolution a less number of times, as indicated by a smaller number for the denominator of the fractional exponent, or root to be sought, as follows:

$$\begin{aligned}\frac{1}{64}^{\frac{1}{3}} &= \frac{1}{4} & \frac{1}{64}^{\frac{1}{2}} &= \frac{1}{8} \\ 0.015625^{\frac{1}{3}} &= 0.25 & 0.015625^{\frac{1}{2}} &= 0.125\end{aligned}$$

This is what is done in Table 1 to the coefficient of C , the competing ability (the multiplier), in order to have E , the earning ability (the product), increased to correspond to the person's ability to compete more successfully in the particular vocation he follows than would be indicated by the coefficient which is given to it from the damage to F , the functional ability of the body, as per ¶ 7, formula (7). Therefore in this consideration of the subject we think we have fully answered the question so often asked: Why is the coefficient of C , the competing ability, in Table 1 increased by evolution and in Table 2 decreased by involution?

¶ 15. It will be observed in this connection that in order to determine the true value of E , the earning ability of a person, that is, in accordance with the actual existing condition of its indispensable elements, these elements must be used as factors and multiplied as per formulas (1), (2), (3), (4), (5), and (6); that when any one of the factors of these formulas becomes 0, the product is 0 also. In this discussion it was shown that this result is mathematically correct, and it was also shown that it is economically correct.

It has been shown (¶ 6) that in normal conditions of health the coefficient of each of the factors of F , the functional ability of the body, is 1, and these multiplied together give a normal coefficient of 1 for F , the functional ability of the person; that when a person is injured, one or more of the factors of F , the functional ability of the body, becomes damaged and, therefore, its coefficient becomes less than 1, and these, being multiplied together in a similar manner as in health, determine the coefficient of F , the remaining functional ability of the body; and although this coefficient must always be less than 1, yet it still remains a composite number. It was also shown in ¶ 7 that the coefficient of F , the functional ability of the body, becomes the coefficient of C , the competing ability of the body, because the indispensable elements of F , the functional ability of the body, are the indispensable elements of C , the competing ability of a person. Therefore, the coefficient of the latter is a composite number and must be treated as such in increasing or diminishing its value. If we wish to diminish its value, we must regard it as a factor and use it as such the number of times desired. In other words, raise it to any given power that will produce the decreased value desired, as illustrated in the examples given, in which the competing ability is damaged to

a greater degree than the functional ability of the person, and also as illustrated by the fraction $\frac{1}{2}$ raised to the 6th power (§ 14).

On the other hand, if we wish to increase the coefficient of the competing ability, we must find a factor or, in other words, a root, of it which multiplied by itself a given number of times will produce it, the root of a fraction always being greater in value than the fraction itself, as per § 14.

The two tables have been arranged in the form of a key, as per § 13, to facilitate their use, the columns being grouped by brackets and designated slight, severe, nearly total, and total, to indicate that the computations in these columns may meet and satisfy the conditions of the competing ability after damage to the functional ability as specified. With this consideration of the formation of the two tables and the directions for using them by means of this key, any one competent to deal with this subject should be able to solve any problem that may come up involving the economic loss from damage to F , the functional ability of the body, from injury or disease, in a manner equitable to all concerned.

Table 3 and Table 4 are complementary tables to Table 1 and Table 2, giving the economic loss on \$1,000 of the economic value of a person for each of these computations. If the decimal point is set one space to the left in Table 3 and Table 4, then the figures as given in the different columns will give the per cent. of loss.

Table 5 explains itself. It may be used as a standard for comparison in determining damages to other parts of the body, as it includes the standard for minor disabilities of the Bureau of Pensions of the United States, namely, a disability equivalent to ankylosis of the wrist joint or the ankle joint, which is designated a loss to factor i of 0.10 ($0.90 i$), and hence, according to § 5, formulas (6) and (2), to F , ($0.90 F$), the functional ability of the body. It also contains the standards for major disabilities of the Bureau of Pensions, namely, a disability equivalent to the loss of a hand at the wrist joint or a foot at the ankle joint. This loss is designated a loss to unit a of 0.20 ($0.80 a$), and hence, according to § 5, formulas (6) and (2), to F ($0.80 F$), the functional ability of the body.

§ 17. In § 4 all the functions of the structures of the entire body are included in the four units a , b , d and g , each of which is divided into three indispensable parts called factors, making

twelve factors for the whole body. Each of these factors is indispensable to the functional ability of a person. In normal conditions of health (§ 6) the coefficient of each of these factors is equal to 1. When the function of any one of them, however, becomes destroyed from injury or disease, its coefficient becomes 0. The quantity between 1 and 0 we may divide into one hundred parts. We must then determine how many of these parts each function of the structures included in a factor represents. In Table 5 we have determined how many of these 100 parts the function of each structure named represents. For instance, the loss of a hand at the wrist joint or a foot at the ankle joint represents one-fifth, or 0.20, of these parts. In Table 7, we have determined that the function of the structures of one ear represents twelve of these parts, or 0.12.

It is not necessary that the functions of all the structures included in one factor should just equal 100 and no more; for instance, the loss of all the functions of the structures included in Table 5. The loss of both arms would be 0.60, and the loss of both legs would be 0.60, making 120 parts. It is self-evident that with the loss of either both arms or both legs a person would have no competing ability, and hence no earning ability.

In factor *w*, according to Table 6 (*a*), Table 7 and Table 8, as far as expressed, there could be a loss of functions as follows:

Both eyes	0.36
Both ears	0.24
Sense of smell.....	0.12
Sense of taste.....	0.06
Making	<hr/> 0.78

The loss of the sense of feeling would make the loss more than 100 per cent. However, with the loss of the function of both eyes and the loss of the function of both ears there would be no competing ability and hence no earning ability.

After we have determined how many parts of the coefficient of each factor the function of each structure shall represent, as in the tables given, we must have a standard of measurement for each of the functions included in the factor. For instance, in the case of the eye we must employ the scientific standard of measurement for normal sight as devised by Snellen, from which I have devised economic standards of measurement to determine the remaining function of the eyes for economic purposes. By these tables we are able to determine the amount of loss of function of sight and the remaining value of the

coefficient of factor w , when the eyes alone are involved, and thereby, with other requisite data and the mathematical formula, to determine the economic loss from damage to the earning ability of a person from damage to the eyes from injury or disease, in a manner equitable to all concerned.

The standards of measurement for the five special senses, Table 6 (*a*), (*a* 1), (*b*), (*b* 1) and (*c*), Table 7 and Table 8, have been constructed on one uniform scale of ten for the scientific standard of measurement, from which scale an economic standard of measurement has been formed consisting of six of this ten scale, namely, from 0.7 to 0.2, inclusive. This economic standard of measurement is formed on the assumption that the first three-tenths of the scientific standard of measurement is not actually necessary for a person to possess in order to follow almost all of the vocations of life successfully. On the other hand, when a person has lost the functions of either one of the special senses to the extent of or below 0.1, that person has not sufficient function for economic purposes, and therefore has sustained a total loss of the function of that special sense.

The scientific standard of measurement for sight is the basis on which Table 6 (*a*) is constructed. It was established by Snellen, a pupil of Donders. At the Seventh International Congress, held in London in 1881, I had the pleasure of going over this subject with Snellen and learning from him how he made the tests and the difficulties he had to overcome in order to determine this scientific standard of measurement. He found by the law of average that the human eye could readily see a letter at twenty feet made on a five-minute angle, and this has been accepted the world over as the scientific standard of measurement for normal sight. The scientific standards of measurement for the other four special senses depends on the same methods for their establishment, and when fully accepted by the weight of authority, they will bear the relations to the economic standard as that indicated in the several tables. For economic purposes, however, it is not difficult to determine the economic standard from accepted scientific methods of measurement for the rest of these special senses sufficiently to designate their loss as slight, severe, and nearly total to total loss of function and thereby ascertain the damage to the functional ability and to the competing ability, and hence the remaining earning ability of the person.

TABLE IX.—ECONOMIC VALUE OF MAN

¶ 18. This table is based on one made by William Farr, M.D., D.C.L., C.B., F.R.S., who spent his life as superintendent of the Registrar-General's office of England, where by his writings on vital statistics and allied subjects he rendered one of the greatest services to his country that ever fell to the lot of man and became an authority on these subjects throughout the whole civilized world. The values in Farr's table were converted into United States money and then changed to a dollar a day basis, or three hundred dollars for the full year. By adopting the dollar a day basis computations can be readily made for a person earning any fractional part of one dollar a day, or three hundred dollars a year. The rate per cent. per annum by which the computations are made is $3\frac{1}{2}$ per cent., compound interest discount.

The present value of the earnings of a person for a prospective future working-life is the sum of these earnings, when discounted at compound interest at the rate per cent. of interest per annum used in the computations for each of the series of years before they will be realized. This makes the interest account of very great economic importance in the problem, because the present value of a sum of money due any number of years hence depends on the rate per cent. of interest per annum and the number of years before it will become due. The present value of such a sum of money is very much less when a high rate of interest is used, for it is the reverse of the amount of a sum of money to be realized at compound interest per annum in the future. The rate of interest here adopted is the one that has been quite universally used in computing values at long terms of interest, though some of the insurance companies now are using a rate of only 3 per cent. per annum for such purposes.

¶ 19. The indispensable elements of the problem of determining the economic value of man are as follows: (1) The earning ability of the individual; (2) the number of years the earning ability is expected to continue according to the condition of the functions of the structures of the body, and the age of the person; (3) the rate per cent. per annum used in the computations of the compound interest discount. We have shown in ¶ 2, that the indispensable elements of the earning ability are (1) the functional ability; (2) the technical ability and (3) the competing ability, and that when these elements

are used as factors and multiplied together they produce the earning ability. This earning ability of the body bears the same relation to the economic value of man as the functional ability of the body bears to the earning ability, namely, the earning ability of the body is a composite quantity and the most important factor in the problem of determining the economic value of man, the same as the functional ability of the body is a composite quantity and the most important factor in the problem of determining the earning ability of man. The economic value of man as here given is made from these indispensable elements used as factors on the basis of one dollar per day, or three hundred dollars per year, at the rate of $3\frac{1}{2}$ per cent. per annum, compound interest discount, so that the economic value of a person can quickly be obtained from what he can earn per day, on an average, for his prospective working life.

Objection is made by those who have given this subject but little thought that the economic value of a person cannot be ascertained. We have just shown that the earning ability of a person is the first indispensable element which must be used as a factor to determine the economic value of that person. We have also pointed out in ¶ 2 the indispensable elements of the earning ability which must be used as factors to determine it. We can determine the economic value of a person only so far as these indispensable elements of the earning ability are manifested in his vocation and the value placed on them as evidenced from the income he receives for his services. We cannot figure on possible prospects of advancements nor change of occupation. We can calculate on the actual conditions of life as they are known to continue to exist, according to the law of average, as defined in the life table, on which has been established life insurance—the first business of the world.

The value of the functions of the body to its possessor cannot be determined, for health, like character, is priceless. Even the possession of health much below any economic value is priceless, and is clung to under all conditions of privations and suffering. This does not affect the purpose of "physical economics," as defined in ¶ 3, for here we have solely to do with the individual's ability to perform certain services and to receive a specified compensation for the remainder of a prospective working life.

Similar practices enter into all business transactions. A person who has no means whatever and could offer no collateral would be refused a loan of money from any person or bank, and he could not obtain money except by reasons which are foreign to the rules of business. A person with good habits and a steady occupation with a specified income would be able to obtain money on that alone, in proportion to his income, other things being equal.

¶ 20. In determining the loss to the functional ability of the body from injury or disease the physician or surgeon who examines the person for that purpose must first examine the functions of the systems and organs and compare the condition in which he finds them with standards of measurement for such systems and organs. By this comparison he determines the character and amount of damage to the functions of each system or organ in any given case. If he finds that each system or organ was in a normal condition before the particular injury in question occurred, then the difference between what he finds and the normal condition represents a fractional loss of each system or organ for that particular injury. If only one system or organ of a factor of a unit is involved, for instance like that of the loss of an eye, in factor w , unit g , which, according to Table 6 (a), is 0.18, we subtract this from 1 and obtain 0.82, which becomes the coefficient of w ($0.82w$). This coefficient of the factor w becomes the coefficient of the unit g ($0.82g$), according to ¶ 5, formula (6), and also the coefficient of F ($0.82F$), the functional ability of the body, according to ¶ 5, formula (2). It also becomes the coefficient of C ($0.82C$), the competing ability of the person, according to ¶ 7, formula (7); and hence we have according to ¶ 8, formula (8), as follows: (11) $0.82F \times 0.82C = E$, the mathematical formula for determining the economic loss from damages to E , the earning ability of a person, for the loss of an eye. If we determine that the 1° slight damage (Table 1, column 10) meets and satisfies the economic loss from the damage to C , the competing ability, in an equitable manner, in the vocation the person follows, then we have the formula complete for computation, according to ¶ 24, formula (10), as follows: (12) $0.82F \times 0.82C^{1/10} = E$. Hence E , the earning ability, is 0.8036 (Table 1, column 10), and the loss is 0.1964, or 19.64 per cent., or \$196.40 on each \$1,000 of the economic value of the person (Table 3, column 10).

If more than one system or organ of a factor is involved in the injury—for instance, if in an accident a person loses an eye and the entire functions of an ear, we would add these two functions together, because they occur in the same factor w , of unit g . We find the sum of these two losses, by the standard of measurement in Table 6 (a), column 5, and Table 7, column 5, to be 0.30. We subtract this from 1 and obtain 0.70, which becomes the coefficient of factor w ($0.70 w$). This also becomes the coefficient of unit g ($0.70 g$), according to ¶ 5, formula (6), and the coefficient of F ($0.70 F$), the functional ability of the body, according to ¶ 5, formula (2). It also becomes the coefficient of C ($0.70 C$), the competing ability of the person, according to ¶ 7, formula (7), and hence we have according to ¶ 8, formula (8), as follows: (13) $0.70 F \times 0.70 C = E$, the statement in the mathematical formula for determining the economic loss from damages to E , the earning ability of a person, for the loss of an eye and the entire function of an ear. If we determine that the 5° slight damage (Table 1, column 5) meets and satisfies the economic loss from the damage to C , the competing ability, in an equitable manner, in the vocation the person follows, then we have the formula complete for computation, according to ¶ 10, formula (10), as follows: (14) $0.70 F \times 0.70 C^{\frac{1}{2}} = E$. Hence E , the earning ability, is 0.6223, and the loss is 0.3777, or 37.77 per cent., or \$377.70 on each \$1,000 of the economic value of the person (Table 3, column 5).

If in an accident one or more of the systems or organs of one or all three of the factors of two or all four of the units of the body are involved in the injury—for instance, if a person should lose the sight of an eye and the sense of smell and the right hand at the wrist joint, we would determine the loss to factor w , according to Table 6 (a) and Table 8 to be 0.30, which subtracted from 1 leaves 0.70, for the coefficient of factor w ($0.70 w$), which, according to ¶ 5, formula (6) becomes the coefficient of g ($0.70 g$). According to Table 5 the loss of a hand at the wrist joint is 0.20, which subtracted from 1 leaves 0.80 for the coefficient of a ($0.80 a$). According to ¶ 5, formula (2), we have as follows: $0.80 a \times b \times d \times 0.70 g = F$. Hence $F = 0.56$ ($0.56 F$) and according to ¶ 7, formula (7), it becomes the coefficient of C , ($0.56 C$) and we have according to ¶ 8, formula (8), as follows: (15) $0.56 F \times 0.56 C = E$. If we determine that the 1° severe damage (Table 1, column 5) meets and satisfies the economic loss from the damage to C ,

the competing ability, in an equitable manner in the vocation the person follows, then we have the formula complete for computation, according to ¶ 10, formula (10), as follows: (16) $0.56 F \times 0.56 C^{1/2} = E$. Hence $E = 0.3136$ (Table 1, column 5), and the loss is 0.6864, or 68.64 per cent., or \$686.40 on each \$1,000 of the economic value of the person (Table 3, column 5).

¶ 21. In 1904 I had the honor of reading a paper before the National Association of the United States Pension Examining Surgeons and members of the Bureau of Pensions of the United States at Atlantic City, N. J., in which I analyzed the methods of giving pensions, showing that the pensions then given were not only based on empiricism, but were full of inconsistencies, absurdities and inequalities, and did not meet the existing conditions of the disabilities of the soldiers and sailors in an equitable manner and do justice to them, and that they therefore should be discarded for scientific methods which would do away with these defects. The result was that ten of the principal pensions were revised in 1905, giving a total increase of \$1,968 for each year.

¶ 22. (1) When the damage to F , the functional ability, is more than 0.80, then the damage to C , the competing ability, is total. (2) When the damage to F , the functional ability, is from 0.50 to 0.80, then the damage to C , the competing ability, comes under the head of nearly total damage. (3) When the damage to F , the functional ability, is from 0.20 to 0.50, then the damage to C , the competing ability, may come under the head of severe damage. (4) When the damage to F , the functional ability, is from 0.01 to 0.19, then the damage to C , the competing ability, may come under the head of slight damage.

¶ 23. Suggestions as to the use of Table 1 and Table 2 and their complementary Table 3 and Table 4, when in any given case of injury to F , the functional ability of the body, it is determined that C , the competing ability, is damaged as follows:

Slight 1°	use $C^{1/10}$	Table 1 column 10 and Table 3 column 10
Slight 2°	use $C^{1/7}$	Table 1 column 9 and Table 3 column 9
Slight 3°	use $C^{1/5}$	Table 1 column 8 and Table 3 column 8
Slight 4°	use $C^{1/4}$	Table 1 column 7 and Table 3 column 7
Slight 5°	use $C^{1/3}$	Table 1 column 6 and Table 3 column 6
Severe 1°	use $C^{1/2}$	Table 1 column 5 and Table 3 column 5
Severe 2°	use C^1	Table 1 column 4 and Table 3 column 4
Severe 3°	use C^2	Table 2 column 2 and Table 4 column 2
Severe 4°	use C^3	Table 2 column 3 and Table 4 column 3
Severe 5°	use C^4	Table 2 column 4 and Table 4 column 4
Severe 6°	use C^5	Table 2 column 5 and Table 4 column 5
Nearly total 1°	use C^6	Table 2 column 6 and Table 4 column 6
Nearly total 2°	use C^7	Table 2 column 7 and Table 4 column 7
Nearly total 3°	use C^8	Table 2 column 8 and Table 4 column 8
Nearly total 4°	use C^9	Table 2 column 9 and Table 4 column 9
Nearly total 5°	use C^{10}	Table 2 column 10 and Table 4 column 10

Total loss C^0 as per paragraph 11.

TABLE 2

Nearly Total										Severe					Loss in F	Slight																					
5°	4°	3°	2°	1°	6°	5°	4°	3°	2°							2°	3°	4°	5°	1°	2°	3°	4°	5°	6°	7°	8°	9°	10°								
Computations made when C is damaged from 2 to 10 degrees more than F																																					
C ¹⁰ E=	C ⁹ E=	C ⁸ E=	C ⁷ E=	C ⁶ E=	C ⁵ E=	C ⁴ E=	C ³ E=	C ² E=	C ¹ E=											F	C	E=	C ^{1.2} E=	C ^{1.4} E=	C ^{1.5} E=	C ^{1.7} E=	C ^{1.9} E=	Computation made when C is damaged to the same degree as F, and when C is damaged to six different degrees less than F									
0.8948	0.9039	0.9129	0.9222	0.9316	0.9411	0.9507	0.9604	0.9702	0.9801	0.99	0.99	0.99	0.99	0.99	0.99	0.99	0.99	0.99	0.99	0.9855	0.9875	0.9888	0.9898	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.989						
0.8902	0.8966	0.9033	0.9104	0.9178	0.9254	0.9332	0.9411	0.9491	0.9572	0.9654	0.9737	0.9821	0.9905	0.9989	1.0073	1.0157	1.0241	1.0325	1.0409	0.9855	0.9875	0.9888	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.98780						
0.8856	0.8922	0.8990	0.9060	0.9131	0.9202	0.9273	0.9344	0.9415	0.9486	0.9557	0.9628	0.9699	0.9770	0.9841	0.9912	0.9983	1.0054	1.0125	1.0196	0.9855	0.9875	0.9888	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.98780						
0.8810	0.8876	0.8943	0.9010	0.9077	0.9144	0.9211	0.9278	0.9345	0.9412	0.9479	0.9546	0.9613	0.9680	0.9747	0.9814	0.9881	0.9948	1.0015	1.0082	0.9855	0.9875	0.9888	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.98780						
0.8764	0.8830	0.8897	0.8964	0.9031	0.9098	0.9165	0.9232	0.9299	0.9366	0.9433	0.9500	0.9567	0.9634	0.9701	0.9768	0.9835	0.9902	0.9969	1.0036	0.9855	0.9875	0.9888	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.98780						
0.8718	0.8784	0.8851	0.8918	0.8985	0.9052	0.9119	0.9186	0.9253	0.9320	0.9387	0.9454	0.9521	0.9588	0.9655	0.9722	0.9789	0.9856	0.9923	0.9990	0.9855	0.9875	0.9888	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.98780						
0.8672	0.8738	0.8805	0.8872	0.8939	0.9006	0.9073	0.9140	0.9207	0.9274	0.9341	0.9408	0.9475	0.9542	0.9609	0.9676	0.9743	0.9810	0.9877	0.9944	0.9855	0.9875	0.9888	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.98780						
0.8626	0.8692	0.8759	0.8826	0.8893	0.8960	0.9027	0.9094	0.9161	0.9228	0.9295	0.9362	0.9429	0.9496	0.9563	0.9630	0.9697	0.9764	0.9831	0.9898	0.9855	0.9875	0.9888	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.98780						
0.8580	0.8646	0.8713	0.8780	0.8847	0.8914	0.8981	0.9048	0.9115	0.9182	0.9249	0.9316	0.9383	0.9450	0.9517	0.9584	0.9651	0.9718	0.9785	0.9852	0.9855	0.9875	0.9888	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.98780						
0.8534	0.8600	0.8667	0.8734	0.8801	0.8868	0.8935	0.9002	0.9069	0.9136	0.9203	0.9270	0.9337	0.9404	0.9471	0.9538	0.9605	0.9672	0.9739	0.9806	0.9855	0.9875	0.9888	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.98780						
0.8488	0.8554	0.8621	0.8688	0.8755	0.8822	0.8889	0.8956	0.9023	0.9090	0.9157	0.9224	0.9291	0.9358	0.9425	0.9492	0.9559	0.9626	0.9693	0.9760	0.9855	0.9875	0.9888	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.98780						
0.8442	0.8508	0.8575	0.8642	0.8709	0.8776	0.8843	0.8910	0.8977	0.9044	0.9111	0.9178	0.9245	0.9312	0.9379	0.9446	0.9513	0.9580	0.9647	0.9714	0.9855	0.9875	0.9888	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.98780						
0.8396	0.8462	0.8529	0.8596	0.8663	0.8730	0.8797	0.8864	0.8931	0.9000	0.9067	0.9134	0.9201	0.9268	0.9335	0.9402	0.9469	0.9536	0.9603	0.9670	0.9855	0.9875	0.9888	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.98780						
0.8350	0.8416	0.8483	0.8550	0.8617	0.8684	0.8751	0.8818	0.8885	0.8952	0.9019	0.9086	0.9153	0.9220	0.9287	0.9354	0.9421	0.9488	0.9555	0.9622	0.9855	0.9875	0.9888	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.98780						
0.8304	0.8370	0.8437	0.8504	0.8571	0.8638	0.8705	0.8772	0.8839	0.8906	0.8973	0.9040	0.9107	0.9174	0.9241	0.9308	0.9375	0.9442	0.9509	0.9576	0.9855	0.9875	0.9888	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.98780						
0.8258	0.8324	0.8391	0.8458	0.8525	0.8592	0.8659	0.8726	0.8793	0.8860	0.8927	0.8994	0.9061	0.9128	0.9195	0.9262	0.9329	0.9396	0.9463	0.9530	0.9855	0.9875	0.9888	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.98780						
0.8212	0.8278	0.8345	0.8412	0.8479	0.8546	0.8613	0.8680	0.8747	0.8814	0.8881	0.8948	0.9015	0.9082	0.9149	0.9216	0.9283	0.9350	0.9417	0.9484	0.9855	0.9875	0.9888	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.98780						
0.8166	0.8232	0.8299	0.8366	0.8433	0.8500	0.8567	0.8634	0.8701	0.8768	0.8835	0.8902	0.8969	0.9036	0.9103	0.9170	0.9237	0.9304	0.9371	0.9438	0.9855	0.9875	0.9888	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.98780						
0.8120	0.8186	0.8253	0.8320	0.8387	0.8454	0.8521	0.8588	0.8655	0.8722	0.8789	0.8856	0.8923	0.8990	0.9057	0.9124	0.9191	0.9258	0.9325	0.9392	0.9855	0.9875	0.9888	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.98780						
0.8074	0.8140	0.8207	0.8274	0.8341	0.8408	0.8475	0.8542	0.8609	0.8676	0.8743	0.8810	0.8877	0.8944	0.9011	0.9078	0.9145	0.9212	0.9279	0.9346	0.9855	0.9875	0.9888	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.98780						
0.8028	0.8094	0.8161	0.8228	0.8295	0.8362	0.8429	0.8496	0.8563	0.8630	0.8697	0.8764	0.8831	0.8898	0.8965	0.9032	0.9099	0.9166	0.9233	0.9300	0.9855	0.9875	0.9888	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.98780						
0.7982	0.8048	0.8115	0.8182	0.8249	0.8316	0.8383	0.8450	0.8517	0.8584	0.8651	0.8718	0.8785	0.8852	0.8919	0.8986	0.9053	0.9120	0.9187	0.9254	0.9855	0.9875	0.9888	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.98780						
0.7936	0.8002	0.8069	0.8136	0.8203	0.8270	0.8337	0.8404	0.8471	0.8538	0.8605	0.8672	0.8739	0.8806	0.8873	0.8940	0.9007	0.9074	0.9141	0.9208	0.9855	0.9875	0.9888	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.98780						
0.7890	0.7956	0.8023	0.8090	0.8157	0.8224	0.8291	0.8358	0.8425	0.8492	0.8559	0.8626	0.8693	0.8760	0.8827	0.8894	0.8961	0.9028	0.9095	0.9162	0.9855	0.9875	0.9888	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.98780						
0.7844	0.7910	0.7977	0.8044	0.8111	0.8178	0.8245	0.8312	0.8379	0.8446	0.8513	0.8580	0.8647	0.8714	0.8781	0.8848	0.8915	0.8982	0.9049	0.9116	0.9855	0.9875	0.9888	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.98780						
0.7798	0.7864	0.7931	0.7998	0.8065	0.8132	0.8199	0.8266	0.8333	0.8400	0.8467	0.8534	0.8601	0.8668	0.8735	0.8802	0.8869	0.8936	0.9003	0.9070	0.9855	0.9875	0.9888	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.98780						
0.7752	0.7818	0.7885	0.7952	0.8019	0.8086	0.8153	0.8220	0.8287	0.8354	0.8421	0.8488	0.8555	0.8622	0.8689	0.8756	0.8823	0.8890	0.8957	0.9024	0.9855	0.9875	0.9888	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.98780						
0.7706	0.7772	0.7839	0.7906	0.7973	0.8040	0.8107	0.8174	0.8241	0.8308	0.8375	0.8442	0.8509	0.8576	0.8643	0.8710	0.8777	0.8844	0.8911	0.8978	0.9855	0.9875	0.9888	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.98780						
0.7660	0.7726	0.7793	0.7860	0.7927	0.7994	0.8061	0.8128	0.8195	0.8262	0.8329	0.8396	0.8463	0.8530	0.8597	0.8664	0.8731	0.8798	0.8865	0.8932	0.9855	0.9875	0.9888	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.98780						
0.7614	0.7680	0.7747	0.7814	0.7881	0.7948	0.8015	0.8082	0.8149	0.8216	0.8283	0.8350	0.8417	0.8484	0.8551	0.8618	0.8685	0.8752	0.8819	0.8886	0.9855	0.9875	0.9888	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.9901	0.98780						
0.7568	0.7634	0.7701	0.7768	0.7835	0.7902	0.7969	0.8036	0.8103	0.8170	0.8237	0.8304	0.8371	0.8438	0.8505	0.8572	0.8639	0.8706	0.8773	0.8840	0.9855	0.9875	0.9888	0.9901	0.9901	0.9901	0.9901	0.9901										

TABLE 2--Continued

Nearly Total					Severe				
5°	4°	3°	2°	1°	6°	5°	4°	3°	2°
10	9	8	7	6	5	4	3	2	
Computations made when C is damaged from 2 to 10 degrees more than F									
C ¹⁰ E=	C ⁹ E=	C ⁸ E=	C ⁷ E=	C ⁶ E=	C ⁵ E=	C ⁴ E=	C ³ E=	C ² E=	
0.0024	0.0042	0.0073	0.0127	0.0219	0.0370	0.0655	0.1131	0.1961	
0.0019	0.0035	0.0062	0.011	0.0194	0.0342	0.055	0.1055	0.1851	
0.0016	0.0029	0.0053	0.0096	0.0172	0.0308	0.051	0.0983	0.1736	
0.0014	0.0026	0.0045	0.0083	0.0151	0.0275	0.0502	0.0914	0.1693	
0.001	0.002	0.0038	0.0071	0.0133	0.0247	0.0458	0.0849	0.1574	
0.0008	0.0016	0.0032	0.0063	0.0117	0.0221	0.0417	0.0788	0.1488	
0.0007	0.0014	0.0027	0.0052	0.0102	0.0197	0.038	0.0731	0.1406	
0.0005	0.0011	0.0022	0.0045	0.0089	0.0175	0.0344	0.0676	0.1326	
0.0004	0.0009	0.0019	0.0039	0.0078	0.0156	0.0312	0.0625	0.1250	
0.0003	0.0007	0.0015	0.0032	0.0067	0.0138	0.0282	0.0570	0.1165	
0.0002	0.0005	0.0012	0.0027	0.0058	0.0121	0.0254	0.0530	0.1105	
0.0001	0.0004	0.001	0.0023	0.0050	0.0107	0.0228	0.0487	0.1038	
0.0001	0.0003	0.0008	0.0019	0.0043	0.0094	0.0205	0.0447	0.0973	
0.0001	0.0003	0.0007	0.0016	0.0036	0.0082	0.0184	0.0409	0.0911	
0.0001	0.0002	0.0005	0.0013	0.0031	0.0072	0.0164	0.0374	0.0851	
0.0001	0.0002	0.0004	0.0011	0.0026	0.0062	0.0146	0.0341	0.0795	
0.0001	0.0001	0.0003	0.0009	0.0022	0.0054	0.013	0.0310	0.0740	
0.0000	0.0001	0.0002	0.0007	0.0019	0.0047	0.0105	0.0282	0.0689	
	0.0000	0.0002	0.0006	0.0016	0.004	0.0102	0.0256	0.064	
		0.0001	0.0005	0.0013	0.0035	0.009	0.0231	0.0538	
		0.0001	0.0004	0.0011	0.0030	0.0079	0.0208	0.0508	
		0.0001	0.0003	0.0009	0.0025	0.0069	0.0187	0.0500	
		0.0002	0.0002	0.0007	0.0021	0.006	0.0167	0.0466	
	0.0001	0.0001	0.0006	0.0018	0.0052	0.0149	0.0428	0.0468	
	0.0001	0.0003	0.0015	0.0045	0.0095	0.0133	0.0393	0.0463	
	0.0001	0.0003	0.0012	0.0038	0.0088	0.0118	0.0359	0.0449	
	0.0000	0.0003	0.0010	0.0033	0.0083	0.0104	0.0327	0.0427	
	0.0000	0.0002	0.0008	0.0062	0.0068	0.0092	0.0297	0.0401	
		0.0001	0.0002	0.0007	0.0057	0.0084	0.0261	0.037	
		0.0001	0.0001	0.0005	0.0045	0.007	0.0243	0.0343	
		0.0001	0.0001	0.0004	0.004	0.007	0.0219	0.0319	
		0.0000	0.0003	0.0003	0.0033	0.0044	0.0032	0.0196	
			0.0002	0.0002	0.0011	0.0045	0.0175	0.0175	
			0.0002	0.0002	0.0009	0.0039	0.0156	0.0156	
			0.0001	0.0001	0.0007	0.0032	0.0138	0.0138	
			0.0001	0.0001	0.0005	0.0027	0.0121	0.0121	
			0.0000	0.0001	0.0004	0.0023	0.0106	0.0106	
			0.0000	0.0000	0.0003	0.0023	0.0019	0.0092	
				0.0000	0.0000	0.0003	0.0016	0.0008	

TABLE 1—Continued

Loss ln F	Severe					Slight									
	2	3	4	5	6	1°	2°	3°	4°	5°	6°	7°	8°	9°	10°
0.42	58	58	58	58	58	0.413	0.4837	0.5075	0.5202	0.5370	0.5492				
0.43	57	57	57	57	57	0.4297	0.4725	0.4950	0.5090	0.5291	0.5386				
0.44	56	56	56	56	56	0.4188	0.4614	0.4844	0.4984	0.5187	0.5296				
0.45	55	55	55	55	55	0.4075	0.4504	0.4735	0.4878	0.5049	0.5181				
0.46	54	54	54	54	54	0.3969	0.4395	0.4627	0.4773	0.4946	0.5076				
0.47	53	53	53	53	53	0.3858	0.4287	0.4520	0.4664	0.4838	0.4971				
0.48	52	52	52	52	52	0.3749	0.4178	0.4414	0.4560	0.4737	0.4872				
0.49	51	51	51	51	51	0.3641	0.4069	0.4309	0.4447	0.4630	0.4768				
0.50	50	50	50	50	50	0.3535	0.3965	0.4205	0.4350	0.4530	0.4665				
0.51	49	49	49	49	49	0.3430	0.3857	0.4096	0.4243	0.4424	0.4561				
0.52	48	48	48	48	48	0.3326	0.3758	0.3993	0.4142	0.4324	0.4459				
0.53	47	47	47	47	47	0.3219	0.3651	0.3886	0.4037	0.4220	0.4356				
0.54	46	46	46	46	46	0.3118	0.3551	0.3785	0.3937	0.4117	0.4255				
0.55	45	45	45	45	45	0.3017	0.3454	0.3681	0.3834	0.4014	0.4153				
0.56	44	44	44	44	44	0.2915	0.3357	0.3581	0.3735	0.3911	0.4052				
0.57	43	43	43	43	43	0.2795	0.3245	0.3478	0.3633	0.3808	0.3951				
0.58	42	42	42	42	42	0.2721	0.3175	0.3398	0.3552	0.3726	0.3871				
0.59	41	41	41	41	41	0.2624	0.3084	0.3301	0.3454	0.3628	0.3767				
0.60	40	40	40	40	40	0.2528	0.2998	0.3214	0.3367	0.3540	0.3684				
0.61	39	39	39	39	39	0.2433	0.2914	0.3129	0.3282	0.3454	0.3598				
0.62	38	38	38	38	38	0.2340	0.2824	0.3039	0.3192	0.3364	0.3508				
0.63	37	37	37	37	37	0.2249	0.2735	0.2950	0.3103	0.3275	0.3419				
0.64	36	36	36	36	36	0.2160	0.2648	0.2863	0.3016	0.3188	0.3332				
0.65	35	35	35	35	35	0.2068	0.2558	0.2773	0.2926	0.3100	0.3244				
0.66	34	34	34	34	34	0.1982	0.2474	0.2689	0.2842	0.3016	0.3160				
0.67	33	33	33	33	33	0.1894	0.2388	0.2603	0.2756	0.2930	0.3074				
0.68	32	32	32	32	32	0.1811	0.2306	0.2521	0.2674	0.2848	0.2992				
0.69	31	31	31	31	31	0.1736	0.2232	0.2447	0.2600	0.2774	0.2918				
0.70	30	30	30	30	30	0.1664	0.2160	0.2375	0.2528	0.2702	0.2846				
0.71	29	29	29	29	29	0.1590	0.2098	0.2314	0.2467	0.2640	0.2784				
0.72	28	28	28	28	28	0.1521	0.2030	0.2246	0.2400	0.2573	0.2717				
0.73	27	27	27	27	27	0.1454	0.1963	0.2179	0.2332	0.2505	0.2649				
0.74	26	26	26	26	26	0.1390	0.1900	0.2116	0.2269	0.2442	0.2586				
0.75	25	25	25	25	25	0.1328	0.1838	0.2054	0.2207	0.2380	0.2524				
0.76	24	24	24	24	24	0.1269	0.1779	0.1995	0.2148	0.2321	0.2465				
0.77	23	23	23	23	23	0.1213	0.1723	0.1939	0.2092	0.2265	0.2409				
0.78	22	22	22	22	22	0.1159	0.1669	0.1885	0.2038	0.2211	0.2355				
0.79	21	21	21	21	21	0.1107	0.1617	0.1833	0.2000	0.2173	0.2317				
0.80	20	20	20	20	20	0.1054	0.1564	0.1780	0.1947	0.2120	0.2264				

TABLE 4—Continued

Nearly Total						Severe						Loss in F	Severe						Slight					
5°	4°	3°	2°	1°		5°	4°	3°	2°	1°			2°	3°	4°	5°	6°	7°	8°	9°	10°			
Loss on \$1,000 when C is damaged from 2 to 10 degrees more than F																								
\$97.60	\$95.80	\$92.70	\$87.30	\$78.10		\$92.10	\$84.50	\$86.90	\$84.90	0.42	.58	.58	.63	.60	\$516.30	\$492.50	\$493.00	.75	.80	\$463.00	\$450.80			
968.10	963.80	963.80	989.00	989.00		963.80	939.90	894.50	814.90	0.43	.57	.57	.60	.60	527.50	504.10	504.10	.49	.50	473.90	461.40			
968.40	967.10	964.70	960.40	962.80		965.00	945.00	901.70	824.40	0.44	.55	.55	.58	.58	538.60	511.00	511.00	.50	.50	484.30	471.40			
968.60	967.40	965.50	961.70	964.10		967.50	949.80	908.60	833.70	0.46	.55	.55	.58	.58	549.60	526.50	526.50	.51	.50	495.10	481.90			
969.00	968.00	966.20	962.30	964.70		969.00	964.20	915.10	824.60	0.46	.54	.54	.55	.55	560.50	537.30	537.30	.52	.50	506.40	492.40			
969.20	968.40	966.80	963.00	965.30		969.30	968.00	921.20	831.00	0.47	.53	.53	.52	.52	571.30	548.00	548.00	.53	.50	517.00	502.90			
969.40	968.80	967.30	964.70	967.00		969.50	968.80	926.90	859.40	0.48	.52	.52	.52	.52	582.00	558.00	558.00	.54	.50	526.30	512.80			
969.50	968.90	967.50	965.00	967.20		969.60	968.60	932.40	867.40	0.49	.51	.51	.51	.51	592.00	569.00	569.00	.55	.50	537.00	523.20			
969.60	969.10	967.80	965.30	967.50		969.70	968.80	937.00	875.00	0.50	.50	.50	.50	.50	601.30	579.50	579.50	.55	.50	547.00	533.50			
969.70	969.30	968.00	965.50	967.70		969.80	968.90	942.40	882.40	0.51	.49	.49	.49	.49	612.90	590.40	590.40	.57	.50	557.00	543.90			
969.80	969.40	968.10	965.60	967.90		969.90	969.00	947.00	889.50	0.52	.48	.48	.48	.48	624.00	600.70	600.70	.58	.50	567.00	554.30			
969.90	969.50	968.20	965.70	968.00		970.00	969.10	951.30	896.20	0.53	.47	.47	.47	.47	634.90	611.40	611.40	.59	.50	578.00	564.40			
969.90	969.60	968.30	965.80	968.10		970.10	969.20	955.30	902.70	0.54	.46	.46	.46	.46	644.90	621.30	621.30	.60	.50	588.30	574.50			
969.90	969.70	968.40	965.90	968.20		970.20	969.30	959.00	908.90	0.55	.45	.45	.45	.45	655.30	631.90	631.90	.61	.50	598.60	584.80			
969.90	969.80	968.50	966.00	968.30		970.30	969.40	962.60	914.90	0.56	.44	.44	.44	.44	665.20	641.90	641.90	.62	.50	608.90	594.90			
969.90	969.90	968.60	966.10	968.40		970.40	969.50	965.00	920.50	0.57	.43	.43	.43	.43	675.00	652.20	652.20	.63	.50	619.10	604.90			
969.90	969.90	968.70	966.20	968.50		970.50	969.60	967.00	926.00	0.58	.42	.42	.42	.42	685.00	662.50	662.50	.64	.50	629.20	614.90			
969.90	969.90	968.80	966.30	968.60		970.60	969.70	968.30	931.00	0.59	.41	.41	.41	.41	695.00	673.00	673.00	.65	.50	640.30	625.30			
969.90	969.90	968.90	966.40	968.70		970.70	969.80	971.40	936.00	0.60	.40	.40	.40	.40	705.00	682.00	682.00	.66	.50	650.30	635.20			
969.90	969.90	968.90	966.40	968.70		970.80	969.90	974.90	940.70	0.61	.39	.39	.39	.39	715.00	691.00	691.00	.67	.50	660.30	645.10			
969.90	969.90	968.90	966.40	968.70		970.90	969.90	978.90	945.20	0.62	.38	.38	.38	.38	724.90	701.00	701.00	.68	.50	670.30	655.00			
969.90	969.90	968.90	966.40	968.70		971.00	970.00	983.30	949.40	0.63	.37	.37	.37	.37	734.90	711.00	711.00	.69	.50	680.30	665.20			
969.90	969.90	968.90	966.40	968.70		971.10	970.10	987.30	953.40	0.64	.36	.36	.36	.36	744.90	720.90	720.90	.70	.50	690.30	675.00			
969.90	969.90	968.90	966.40	968.70		971.20	970.20	991.30	957.20	0.65	.35	.35	.35	.35	754.90	730.90	730.90	.71	.50	700.30	685.00			
969.90	969.90	968.90	966.40	968.70		971.30	970.30	995.30	960.70	0.66	.34	.34	.34	.34	764.90	740.90	740.90	.72	.50	710.30	694.70			
969.90	969.90	968.90	966.40	968.70		971.40	970.40	999.30	964.10	0.67	.33	.33	.33	.33	774.90	749.90	749.90	.73	.50	720.30	704.70			
969.90	969.90	968.90	966.40	968.70		971.50	970.50	1003.30	967.30	0.68	.32	.32	.32	.32	784.90	759.90	759.90	.74	.50	730.30	714.60			
969.90	969.90	968.90	966.40	968.70		971.60	970.60	1007.30	970.30	0.69	.31	.31	.31	.31	794.90	769.90	769.90	.75	.50	740.30	724.50			
969.90	969.90	968.90	966.40	968.70		971.70	970.70	1011.30	973.00	0.70	.30	.30	.30	.30	804.90	779.90	779.90	.76	.50	750.30	734.40			
969.90	969.90	968.90	966.40	968.70		971.80	970.80	1015.30	975.70	0.71	.29	.29	.29	.29	814.90	789.90	789.90	.77	.50	760.30	744.30			
969.90	969.90	968.90	966.40	968.70		971.90	970.90	1019.30	978.10	0.72	.28	.28	.28	.28	824.90	799.90	799.90	.78	.50	770.30	754.20			
969.90	969.90	968.90	966.40	968.70		972.00	971.00	1023.30	980.40	0.73	.27	.27	.27	.27	834.90	809.90	809.90	.79	.50	780.30	764.10			
969.90	969.90	968.90	966.40	968.70		972.10	971.10	1027.30	982.50	0.74	.25	.25	.25	.25	844.90	819.90	819.90	.80	.50	790.30	774.00			
969.90	969.90	968.90	966.40	968.70		972.20	971.20	1031.30	984.40	0.75	.25	.25	.25	.25	854.90	829.90	829.90	.81	.50	800.30	783.90			
969.90	969.90	968.90	966.40	968.70		972.30	971.30	1035.30	986.20	0.76	.24	.24	.24	.24	864.90	839.90	839.90	.82	.50	810.30	793.80			
969.90	969.90	968.90	966.40	968.70		972.40	971.40	1039.30	987.90	0.77	.23	.23	.23	.23	874.90	849.90	849.90	.83	.50	820.30	803.70			
969.90	969.90	968.90	966.40	968.70		972.50	971.50	1043.30	989.40	0.78	.22	.22	.22	.22	884.90	859.90	859.90	.84	.50	830.30	813.50			
969.90	969.90	968.90	966.40	968.70		972.60	971.60	1047.30	990.80	0.79	.22	.22	.22	.22	894.90	869.90	869.90	.85	.50	840.30	823.40			
969.90	969.90	968.90	966.40	968.70		972.70	971.70	1051.30	992.00	0.80	.20	.20	.20	.20	904.90	879.90	879.90	.86	.50	850.30	833.30			
969.90	969.90	968.90	966.40	968.70		972.80	971.80	1055.30	993.00						914.90	889.90	889.90			860.30	843.20			
969.90	969.90	968.90	966.40	968.70		972.90	971.90	1059.30	994.00						924.90	899.90	899.90			870.30	853.10			
969.90	969.90	968.90	966.40	968.70		973.00	972.00	1063.30	995.00						934.90	909.90	909.90			880.30	863.00			
969.90	969.90	968.90	966.40	968.70		973.10	972.10	1067.30	996.00						944.90	919.90	919.90			890.30	872.90			
969.90	969.90	968.90	966.40	968.70		973.20	972.20	1071.30	997.00						954.90	929.90	929.90			900.30	882.80			
969.90	969.90	968.90	966.40	968.70		973.30	972.30	1075.30	998.00						964.90	939.90	939.90			910.30	892.70			
969.90	969.90	968.90	966.40	968.70		973.40	972.40	1079.30	999.00						974.90	949.90	949.90			920.30	902.60			
969.90	969.90	968.90	966.40	968.70		973.50	972.50	1083.30	1000.00						984.90	959.90	959.90			930.30	912.50			
969.90	969.90	968.90	966.40	968.70		973.60	972.60	1087.30							994.90	969.90	969.90			940.30	922.40			
969.90	969.90	968.90	966.40	968.70		973.70	972.70	1091.30							1004.90	979.90	979.90			950.30	932.30			
969.90	969.90	968.90	966.40	968.70		973.80	972.80	1095.30								989.90	989.90			960.30	942.20			
969.90	969.90	968.90	966.40	968.70		973.90	972.90	1099.30								999.90	999.90			970.30	952.10			
969.90	969.90	968.90	966.40	968.70		974.00	973.00	1103.30								1009.90	1009.90			980.30	962.00			
969.90	969.90	968.90	966.40	968.70		974.10	973.10	1107.30												990.30	971.90			
969.90	969.90	968.90	966.40	968.70		974.20	973.20	1111.30												1000.30	981.80			
969.90	969.90	968.90	966.40	968.70		974.30	973.30	1115.30													991.70			
969.90	969.90	968.90	966.40	968.70		974.40	973.40	1119.30													1001.60			
969.90	969.90	968.90	966.40	968.70		974.50	973.50	1123.30													1011.50			
969.90	969.90	968.90	966.40	968.70		974.60	973.60	1127.30													1021.40			
969.90	969.90	968.90	966.40	968.70		974.70	973.70	1131.30													1031.30			
969.90	969.90	968.90	966.40	968.70		974.80	973.80	1135.30													1041.20			
969.90	969.90	968.90	966.40	968.70		974.90	973.90	1139.30		</														

TABLE 5.—STANDARD OF MEASUREMENT FOR DETERMINING DAMAGES TO F (a) OF THE BODY

Loss in F (a) in ankylosis of		Loss in F (a) in Amputation of	
Fingers	Toes	Fingers	Toes
Little0.01	Little0.005	Little0.01	Little0.005
Ring0.02	Fourth0.005	Ring0.02	Fourth0.005
Middle0.02	Middle0.005	Middle0.03	Middle0.01
Index0.02	Second0.005	Index0.04	Second0.01
Thumb0.03	Big0.01	Thumb0.05	Big0.02
Wrist0.10	Ankle0.10	Hand0.20	Foot0.20
Elbow0.15	Knee0.15	Forearm0.25	Leg0.25
Shoulder0.15	Hip0.15	Arm0.30	Thigh0.30

¶ 24. Table 6 (a).—Standard of measurement for the purpose of determining the economic loss from damages to *F*, the functional ability of the body, for the partial or complete loss of two or all three of the functions of one eye (unit *g*, factor *w*) :

Degree of Disability	Scientific Standard from	Economic Standard	Loss to	Loss to F from
Slight loss of two or all three of the functions of one eye	{ 0.7 to 0.633 0.633 to 0.566 0.566 to 0.5	{ 1° from 9/9 to 8/9 2° from 8/8 to 7/9 3° from 7/9 to 6/9	{ 1/9 2/9 3/9	{ 0.00 to 0.02 (1) 0.02 to 0.04 (2) 0.04 to 0.06 (3)
Severe loss of two or all three of the functions of one eye	{ 0.5 to 0.433 0.433 to 0.366 0.366 to 0.3	{ 1° from 6/9 to 5/9 2° from 5/9 to 4/9 3° from 4/9 to 3/9	{ 4/9 5/9 6/9	{ 0.06 to 0.08 (4) 0.08 to 0.10 (5) 0.10 to 0.12 (6)
Nearly total to total loss of two or all three of the functions of one eye.....	{ 0.3 to 0.233 0.233 to 0.166 0.166 to 0.1	{ 1° from 3/9 to 2/9 2° from 2/9 to 1/9 3° from 1/9 to 0	{ 7/9 8/9 9/9	{ 0.12 to 0.14 (7) 0.14 to 0.16 (8) 0.16 to 0.18 (9)

In case of the loss of two or all three of the functions of one eye to 0.1 or less of normal, the loss to the functional ability of the eye for economic purposes is total, and the loss to *F*, the functional ability of the body, for economic purposes would be 0.18 (10).

¶ 25. Table 6 (a).—This table is the standard of measurement for the partial or complete loss of two or all three of the indispensable functions of vision of one eye, namely, central acuity of sight, the field of vision, and the muscular functions for economic purposes.

¶ 26. In case of the loss of two of the indispensable functions of vision, namely, the central acuity of sight and the field of vision, which occurs in blindness of one eye, the loss to the functional ability of the eye for economic purposes would be total, even though the muscular functions were normal, and the loss to *F*, the functional ability of the body, would be 0.18.

¶ 27. In case of the loss of the central acuity of sight with the loss of the function of one or more of the muscles from paralysis, causing the eye to become crossed, the eye would not

have to be excluded, because there would be no double vision, and the function of the field of vision would be useful in any vocation, hence the competing ability would be greater than though the eye were excluded from taking part in vision, nevertheless, as the eye has lost its central acuity of sight and become paralyzed in its movements, the loss to the functional ability should be considered total, and to F , the functional ability of the body, 0.18.

¶ 28. In case of the loss of the field of vision of one eye with the loss of the functions of one or more of the muscles, causing the eye to become crossed, the eye would have to be excluded on account of the annoying double vision, hence there would be a total loss of the functional ability of the eye and the loss to F , the functional ability of the body, would be 0.18.

¶ 29. Table 6 (*a1*).—Standard of measurement for the purpose of determining the economic loss from damages to F , the functional ability of the body, for the partial or complete loss of the function of central acuity of sight of one eye, the field of vision and muscular function of the eye being normal (unit g , factor w).

Degree of Disability	Scientific Standard from	Economic Standard from	Loss to	Loss to F from
Slight loss of the central acuity of sight	{ 0.7 to 0.633 0.633 to 0.566 0.566 to 0.5	{ 1° from 9/9 to 8/9 2° from 8/9 to 7/9 3° from 7/9 to 6/9	{ 1/9 2/9 3/9	{ 0.00 to 0.01 (1) 0.01 to 0.02 (2) 0.02 to 0.03 (3)
Severe loss of the central acuity of sight	{ 0.5 to 0.433 0.433 to 0.366 0.366 to 0.3	{ 1° from 6/9 to 5/9 2° from 5/9 to 4/9 3° from 4/9 to 3/9	{ 4/9 5/9 6/9	{ 0.03 to 0.04 (4) 0.04 to 0.05 (5) 0.05 to 0.06 (6)
Nearly total to total loss of central acuity of sight ...	{ 0.3 to 0.233 0.233 to 0.167 0.167 to 0.1	{ 1° from 3/9 to 2/9 2° from 2/9 to 1/9 3° from 1/9 to 0	{ 7/9 8/9 9/9	{ 0.06 to 0.07 (7) 0.07 to 0.08 (8) 0.08 to 0.09 (9)

In case of the loss of the central acuity of sight to 0.1 or less of normal, there is a loss of one-half of the functional ability of the eye and the loss to F , the functional ability of the body, for economic purposes would be 0.09 (10).

¶ 30. Table 6 (*a1*) is the standard of measurement for the partial or complete loss of only one of the indispensable functions of vision of one eye, namely, the central acuity of sight according to scientific and economic standards of measurement, the field of vision and the muscular functions being normal.

¶ 31. In case of the loss of the central acuity of sight from 0.7 to 0.5 of normal, the loss to the functional ability of the eye is slight, and to F , the functional ability of the body, from 0 to 0.03.

¶ 32. In case of the loss of the central acuity of sight from 0.5 to 0.3 of normal, the loss to this functional ability of the eye is severe, and to F , the functional ability of the body, from 0.03 to 0.06.

¶ 33. In case of the loss of the central acuity of sight from 0.3 to 0.1 of normal, the loss to this functional ability of the eye would be nearly total to a total loss, and to F , the functional ability of the body, from 0.06 to 0.09.

¶ 34. In case of the loss of the central acuity of sight to 0.1 or less of normal, the loss to this functional ability of the eye is total, and to F , the functional ability of the body would be 0.09.

¶ 35. Table 6 (*b*).—Standard of measurement for determining the economic loss from damages to F , the functional ability of the body, for the partial or complete loss of the function of the field of vision of one eye from concentric or its equivalent in irregular contraction, the central acuity of sight and the muscular functions being normal (unit g , factor w).

Degree of Disability	Scientific Standard from	Economic Standard	Loss to	Loss to F from
Slight loss of the field of vision	150° to 140°	1° from 9/9 to 8/9	1/9	0.00 to 0.01 (1)
	140° to 130°	2° from 8/9 to 7/9	2/9	0.01 to 0.02 (2)
	130° to 120°	3° from 7/9 to 6/9	3/9	0.02 to 0.03 (3)
Severe loss of the field of vision	120° to 100°	1° from 6/9 to 5/9	4/9	0.03 to 0.04 (4)
	100° to 80°	2° from 5/9 to 4/9	5/9	0.04 to 0.05 (5)
	80° to 60°	3° from 4/9 to 3/9	6/9	0.05 to 0.06 (6)
Nearly total to total loss of the field of vision	60° to 40°	1° from 3/9 to 2/9	7/9	0.06 to 0.07 (7)
	40° to 20°	2° from 2/9 to 1/9	8/9	0.07 to 0.08 (8)
	20° to 10°	3° from 1/9 to 0	9/9	0.08 to 0.09 (9)

In case of the loss in the field of vision of one eye to within 5° of the point of fixation, the central acuity of sight and the muscular functions of the eye being normal, there is a loss of one-half of the functional ability of the eye, and the loss to F , the functional ability of the body for economic purposes, would be 0.09 (10).

¶ 36. Table 6 (*b*).—Standard of measurement for only one of the indispensable functions of vision of one eye, namely, the field of vision, the central acuity of sight and the muscular functions of the eye being normal.

¶ 37. In case of the loss in the field of vision from 150° to 120° from concentric or its equivalent in irregular contraction, the central acuity of sight and the muscular function of the eye being normal, the loss to the functional ability of the eye would be slight, and the loss to F , the functional ability of the body, would be from 0 to 0.03.

¶ 38. In case of the loss in the field of vision from 120° to 60° from concentric or its equivalent in irregular contraction, the central acuity of sight and the muscular functions of the eye being normal, the loss to this functional ability of the eye would be severe, and the loss to F , the functional ability of the body, would be from 0.03 to 0.06.

¶ 39. In case of the loss in the field of vision from 60° to 10° from concentric or its equivalent in irregular contraction, the central acuity of sight and the muscular functions of the eye being normal, the loss to this functional ability of the eye would be nearly total to a total loss, and the loss to F , the functional ability of the body, would be 0.06 to 0.09.

¶ 40. In the case of the loss in the field of vision of one eye to within 5° of the point of fixation, the central acuity of sight and the muscular function being normal, the loss to this functional ability of the eye is total, and the loss to F , the functional ability of the body, would be 0.09.

¶ 41. In case of the total loss of the temporal field of vision of one eye, the loss to F , the functional ability of the body, would be 0.09.

¶ 42. In case of the total loss of the nasal field of vision of one eye, there is no loss in the area of the field of vision, only a loss of the normal overlapping of the nasal field of vision on the temporal field of vision, hence the loss to the functional ability of the eye is slight, and the loss to F , the functional ability of the body, would be from 0 to 0.3.*

¶ 43. Table 6 (b 1).—Standard of measurement for the purpose of determining the economic loss from damages to F , the functional ability of the body, for the partial or complete loss of the functions of the field of vision of both eyes by concentric or its equivalent in irregular contractions (unit g , factor w).

Degree of Disability	Scientific Standard from	Economic Standard	Loss to	Loss to F from
Slight loss of the field of vision	$\left\{ \begin{array}{l} 180^\circ \text{ to } 160^\circ \\ 160^\circ \text{ to } 140^\circ \\ 140^\circ \text{ to } 120^\circ \end{array} \right.$	$\left\{ \begin{array}{l} 1^\circ \text{ from } 9/9 \text{ to } 8/9 \\ 2^\circ \text{ from } 8/9 \text{ to } 7/9 \\ 3^\circ \text{ from } 7/9 \text{ to } 6/9 \end{array} \right.$	$\left\{ \begin{array}{l} 1/9 \\ 2/9 \\ 3/9 \end{array} \right.$	$\left\{ \begin{array}{l} 0.00 \text{ to } 0.04 \text{ (1)} \\ 0.04 \text{ to } 0.08 \text{ (2)} \\ 0.08 \text{ to } 0.12 \text{ (3)} \end{array} \right.$
Severe loss to the field of vision	$\left\{ \begin{array}{l} 120^\circ \text{ to } 100^\circ \\ 100^\circ \text{ to } 80^\circ \\ 80^\circ \text{ to } 60^\circ \end{array} \right.$	$\left\{ \begin{array}{l} 1^\circ \text{ from } 6/9 \text{ to } 5/9 \\ 2^\circ \text{ from } 5/9 \text{ to } 4/9 \\ 3^\circ \text{ from } 4/9 \text{ to } 3/9 \end{array} \right.$	$\left\{ \begin{array}{l} 4/9 \\ 5/9 \\ 6/9 \end{array} \right.$	$\left\{ \begin{array}{l} 0.12 \text{ to } 0.16 \text{ (4)} \\ 0.16 \text{ to } 0.20 \text{ (5)} \\ 0.20 \text{ to } 0.24 \text{ (6)} \end{array} \right.$
Nearly total to total loss of the field of vision	$\left\{ \begin{array}{l} 60^\circ \text{ to } 40^\circ \\ 40^\circ \text{ to } 20^\circ \\ 20^\circ \text{ to } 10^\circ \end{array} \right.$	$\left\{ \begin{array}{l} 1^\circ \text{ from } 3/9 \text{ to } 2/9 \\ 2^\circ \text{ from } 2/9 \text{ to } 1/9 \\ 3^\circ \text{ from } 1/9 \text{ to } 0 \end{array} \right.$	$\left\{ \begin{array}{l} 7/9 \\ 8/9 \\ 9/9 \end{array} \right.$	$\left\{ \begin{array}{l} 0.24 \text{ to } 0.28 \text{ (7)} \\ 0.28 \text{ to } 0.32 \text{ (8)} \\ 0.32 \text{ to } 0.36 \text{ (9)} \end{array} \right.$

* Magnus does not consider the loss of one nasal field, or even both nasal fields, of any economic loss when both temporal fields are normal, but as the loss of the normal overlapping of the nasal upon the temporal field weakens the central field of vision, we consider that a person has sustained a slight loss of the functions of an eye when he has lost one nasal field or both nasal fields of vision under these conditions.

In case of the loss of the field of vision of both eyes to within 5° of the point of fixation, the loss to the functional ability of the eyes is total, and the loss to F , the functional ability of the body, would be 0.36 (10).

¶ 44. Table 6 (b 1).—The remaining field of vision is the same in area in Table 6 (b) as in Table 6 (b-1), beginning at severe loss of the field of vision, but in the former it is a single field of vision that is lost while in the latter it is a double field of vision that is lost.

¶ 45. For the partial or complete loss of the field of vision of a one-eyed person the standard of measurement given in Table 6 (b 1) should be used instead of that given in Table 6 (b).

¶ 46. Severe loss of the field of vision of both eyes, namely, from 120° to 60° , would damage the competing ability of a person to a severe degree, and might in some vocations damage it to nearly a total or to a total degree.

¶ 47. Nearly total to total loss of the field of vision of both eyes would damage the competing ability of a person to nearly a total or to a total degree. Hence, such a person would be unable to follow any vocation successfully and therefore would really have no earning ability and hence no economic value. If in such a case this loss was due to an injury, which would be extremely rare, then the indemnity should be for total disability.

¶ 48. If there were total loss to the temporal field of vision of both eyes, the loss to the functional ability of the eyes would be equal to that of the loss of an eye, and hence the loss to F , the functional ability of the body, would be 0.18.

¶ 49. If there were total loss of the temporal field of vision of one eye with total loss of the nasal field of the other eye (homonymous hemianopsia, see chart, figures 9 and 10), the loss to the functional ability of the eyes would be equal to that of the functional ability of one eye, and hence the loss to F , the functional ability of the body, would be 0.18.

¶ 50. If there is a total loss of the nasal field of vision of one eye only, there is no loss in the area of the binocular field, only a loss of the normal overlapping of this field in the temporal field of vision of the other eye, and therefore the loss to the functional ability of the eye would be slight and hence the loss to F , the functional ability of the body, would be from 0 to 0.03.

¶ 51. If there is a total loss of the nasal field of vision of both eyes, there is no loss in the area of the binocular field

of vision, only a loss of the normal overlapping of each nasal field in the corresponding temporal field, hence the loss to the functional ability of the eyes is slight and the loss to F , the functional ability of the body, would be from 0 to 0.06.

¶ 52. If there is a loss of the temporal field of vision, for instance, of the left eye, with loss of the nasal field of vision of the right eye, nothing is seen to the left of a vertical line, but the field of vision to the right of the vertical line is normal. This was named left homonymous hemianopsia, that is, left half-sight, thus naming the functions of the eye retained.

¶ 53. In case of hemianopsia,* right or left superior or inferior, the loss to the functional ability of the eyes will be equal to the loss of the function of one eye, and hence the loss to F , the functional ability of the body, would be 0.18.

¶ 54. The loss to F , the functional ability of the body, for the loss in the field of vision considered in addition to the tables, may be gathered together as follows:

1. The loss of the temporal field of vision of one eye, 0.09.
2. The loss of the temporal field of vision of both eyes, 0.18.
3. The loss of the temporal field of vision of the left eye with loss of the nasal field of vision of the right eye, known as left homonymous hemianopsia, 0.18.
4. The loss of the temporal field of vision of the right eye with loss of the nasal field of the left eye, known as right homonymous hemianopsia, 0.18.
5. The loss of the superior field of vision of both eyes, known as superior hemianopsia, 0.18.
6. The loss of the inferior field of vision of both eyes, known as inferior hemianopsia, 0.18.
7. The loss of one nasal field of vision only, 0 to 0.03.
8. The loss of both nasal fields of vision, 0 to 0.06.

¶ 55. Relative to homonymous hemianopsia, in all cases that have come under our observation during the past forty years the competing ability of the persons has been nearly totally damaged, so much so that none of them has had sufficient earning ability to take care of himself, and hence, he has had no economic value.

* We have had cases in which there was a loss of one fourth of the field of vision in each lower half, but it could not in any case be traced to an injury. The loss, however, to the functional ability of the eyes would be similar to that of complete hemianopsia, and hence the loss to F , the functional ability of the body would be 0.18.

¶ 56. Table 6 (c).—Standard of measurement for the purpose of determining the economic loss from damages to F , the functional ability of the body, for the partial or complete loss of the functions of one or more of the muscles of one eye (unit g , factor w).

Degree of Disability	Scientific Standard	Economic Standard	Loss to	Loss to F from
Slight loss of the functions of one or more of the muscles	This consists of the scientific standard of measurement for orthophoria, for orthotropia, for cyclophoria, for field of fusion, for field of fixation, for range of accommodation, and for paresis, or paralysis of a muscle or muscles.	1° from 9/9 to 8/9	1/9	0.99 to 0.02 (1)
		2° from 8/9 to 7/9	2/9	0.02 to 0.04 (2)
		3° from 7/9 to 6/9	3/9	0.04 to 0.06 (3)
Severe loss of the functions of one or more of the muscles		1° from 6/9 to 5/9	4/9	0.06 to 0.08 (4)
		2° from 5/9 to 4/9	5/9	0.08 to 0.10 (5)
		3° from 4/9 to 3/9	6/9	0.10 to 0.12 (6)
Nearly total to total loss of the functions of one or more of the muscles.....		1° from 3/9 to 2/9	7/9	0.12 to 0.14 (7)
		2° from 2/9 to 1/9	8/9	0.14 to 0.16 (8)
		3° from 1/9 to 0	9/9	0.16 to 0.18 (9)

In case of the loss of the function of one or more of the muscles of one eye necessitating the exclusion of the eye, there is a total loss of the functional ability of that eye, and hence the loss to F , the functional ability of the body, would be 0.18 (10).

¶ 57. Table 6 (c) includes the functions of all the muscles connected with the visual act, namely, (1) the internal rectus, (2) superior rectus, (3) inferior rectus, (4) inferior oblique, (5) the levator of the upper lid, (6) the iris and ciliary muscles, all supplied by the third nerve; (7) the superior oblique, supplied by the fourth nerve; (8) the external rectus, supplied by the sixth nerve, and (9) the orbicularis muscles of the lid, supplied by the seventh nerve.

¶ 58. This table for the partial or complete loss of one or more of the muscular functions of one eye is not so complete nor so specific as the preceding tables for the reason that the nature of the functions of the muscles of the eyes do not permit it, and therefore supplements are offered for the purpose of assisting the expert in arriving at more definite conclusions as to the loss of the functional ability of the eye in a given case, as follows:

¶ 59. When one eye is necessarily and permanently prevented from taking part in vision from paralysis of any one or all of the muscles connected with the act of vision, then the loss to the functional ability of the eye is the same as the loss of the entire vision or the loss of the eye and hence the loss to F , the functional ability of the body, would be 0.18.

¶ 60. When the function of one eye is lost and there is paralysis of one or more of the muscles of the remaining eye,

there should be added for each muscle paralyzed one-ninth of the economic standard as given in Table 6 (c), namely 0.02 as follows:

For the paralysis of one of the muscles of the remaining eye $0.18 + 0.02 = 0.20$ (1).

For the paralysis of two of the muscles of the remaining eye $0.18 + 0.04 = 0.22$ (2).

For the paralysis of three of the muscles of the remaining eye $0.18 + 0.06 = 0.24$ (3).

For the paralysis of four of the muscles of the remaining eye $0.18 + 0.08 = 0.26$ (4).

For the paralysis of five of the muscles of the remaining eye $0.18 + 0.10 = 0.28$ (5).

For the paralysis of six of the muscles of the remaining eye $0.18 + 0.12 = 0.30$ (6).

For the paralysis of seven of the muscles of the remaining eye $0.18 + 0.14 = 0.32$ (7).

For the paralysis of eight of the muscles of the remaining eye $0.18 + 0.16 = 0.34$ (8).

For the paralysis of nine of the muscles of the remaining eye $0.18 + 0.18 = 0.36$ (9).

¶ 61. Table 7.—Standard of measurement for the purpose of determining the economic loss from damages to F , the functional ability of the body, for the partial or complete loss of the function of hearing in one ear (unit g , factor w).

Degree of Disability	Scientific Standard	Economic Standard	Loss to	Loss to F
For slight loss of the function of hearing in one ear	$\left\{ \begin{array}{l} 1^\circ \text{ from } 0.7 \text{ to } 0.6 \\ 2^\circ \text{ from } 0.6 \text{ to } 0.5 \end{array} \right.$	$\left\{ \begin{array}{l} \text{from } 6/6 \text{ to } 5/6 \\ \text{from } 5/6 \text{ to } 4/6 \end{array} \right.$	$\left\{ \begin{array}{l} 1/6 \\ 2/6 \end{array} \right.$	$\left\{ \begin{array}{l} \text{from } 0.00 \text{ to } 0.02 \\ \text{from } 0.02 \text{ to } 0.04 \end{array} \right.$
For severe loss of the function of hearing in one ear. . . .	$\left\{ \begin{array}{l} 1^\circ \text{ from } 0.5 \text{ to } 0.4 \\ 2^\circ \text{ from } 0.4 \text{ to } 0.3 \end{array} \right.$	$\left\{ \begin{array}{l} \text{from } 4/6 \text{ to } 3/6 \\ \text{from } 3/6 \text{ to } 2/6 \end{array} \right.$	$\left\{ \begin{array}{l} 3/6 \\ 4/6 \end{array} \right.$	$\left\{ \begin{array}{l} \text{from } 0.04 \text{ to } 0.06 \\ \text{from } 0.06 \text{ to } 0.08 \end{array} \right.$
For nearly total loss of the function of hearing in one ear	$\left\{ \begin{array}{l} 1^\circ \text{ from } 0.3 \text{ to } 0.2 \\ 2^\circ \text{ from } 0.2 \text{ to } 0.1 \end{array} \right.$	$\left\{ \begin{array}{l} \text{from } 2/6 \text{ to } 1/6 \\ \text{from } 1/6 \text{ to } 0 \end{array} \right.$	$\left\{ \begin{array}{l} 5/6 \\ 6/6 \end{array} \right.$	$\left\{ \begin{array}{l} \text{from } 0.08 \text{ to } 0.10 \\ \text{from } 0.10 \text{ to } 0.12 \end{array} \right.$
For total loss of the function of hearing in one ear, 0.12				

¶ 62. Table 8.—Standard of measurement for the purpose of determining the economic loss from damages to F , the functional ability of the body, for the partial or complete loss of the function of smell.

Degree of Disability	Scientific Standard	Economic Standard	Loss to	Loss to F
For slight loss of the function of smell	$\left\{ \begin{array}{l} 1^\circ \text{ from } 0.7 \text{ to } 0.6 \\ 2^\circ \text{ from } 0.6 \text{ to } 0.5 \end{array} \right.$	$\left\{ \begin{array}{l} \text{from } 6/6 \text{ to } 5/6 \\ \text{from } 5/6 \text{ to } 4/6 \end{array} \right.$	$\left\{ \begin{array}{l} 1/6 \\ 2/6 \end{array} \right.$	$\left\{ \begin{array}{l} \text{from } 0.00 \text{ to } 0.02 \\ \text{from } 0.02 \text{ to } 0.04 \end{array} \right.$
For severe loss of the function of smell	$\left\{ \begin{array}{l} 1^\circ \text{ from } 0.5 \text{ to } 0.4 \\ 2^\circ \text{ from } 0.4 \text{ to } 0.3 \end{array} \right.$	$\left\{ \begin{array}{l} \text{from } 4/6 \text{ to } 3/6 \\ \text{from } 3/6 \text{ to } 2/6 \end{array} \right.$	$\left\{ \begin{array}{l} 3/6 \\ 4/6 \end{array} \right.$	$\left\{ \begin{array}{l} \text{from } 0.04 \text{ to } 0.06 \\ \text{from } 0.06 \text{ to } 0.08 \end{array} \right.$
For nearly total loss of the function of smell	$\left\{ \begin{array}{l} 1^\circ \text{ from } 0.3 \text{ to } 0.2 \\ 2^\circ \text{ from } 0.2 \text{ to } 0.1 \end{array} \right.$	$\left\{ \begin{array}{l} \text{from } 2/6 \text{ to } 1/6 \\ \text{from } 1/6 \text{ to } 0 \end{array} \right.$	$\left\{ \begin{array}{l} 5/6 \\ 6/6 \end{array} \right.$	$\left\{ \begin{array}{l} \text{from } 0.08 \text{ to } 0.10 \\ \text{from } 0.10 \text{ to } 0.12 \end{array} \right.$
For total loss of the function of smell, 0.12.				

For the partial or complete loss of the function of taste:

Degree of Disability	Scientific Standard	Economic Standard	Loss to	Loss to F
For slight loss of the function of taste	{ 1° from 0.7 to 0.6 2° from 0.6 to 0.5	{ from 6/6 to 5/6 from 5/6 to 4/6	{ 1/6 2/6	{ from 0.00 to 0.01 from 0.01 to 0.02
For severe loss of the function of taste	{ 1° from 0.5 to 0.4 2° from 0.4 to 0.3	{ from 4/6 to 3/6 from 3/6 to 2/6	{ 3/6 4/6	{ from 0.02 to 0.03 from 0.03 to 0.04
For nearly total loss of the function of taste	{ 1° from 0.3 to 0.2 2° from 0.2 to 0.1	{ from 2/6 to 1/6 from 1/6 to 0	{ 5/6 6/6	{ from 0.04 to 0.05 from 0.05 to 0.06
For total loss of the function of taste, 0.06.				

For the partial or complete loss of the function of feeling:

Degree of Disability	Scientific Standard	Economic Standard	Loss	Loss to F
For slight loss of the function of feeling	{ 1° from 0.7 to 0.6 2° from 0.6 to 0.5	{ from 6/5 to 5/6 from 5/6 to 4/6	{ 1/6 2/6	Depends upon the extent of the area involved and the functions of the part, or parts, which have lost the sense of feeling.
For severe loss of the function of feeling	{ 1° from 0.5 to 0.4 2° from 0.4 to 0.3	{ from 4/6 to 3/6 from 3/6 to 2/6	{ 3/6 4/6	
For nearly total loss of the function of feeling	{ 1° from 0.3 to 0.2 2° from 0.2 to 0.1	{ from 2/6 to 1/6 from 1/6 to 0	{ 5/6 6/6	

Total loss of feeling, do.

¶ 63. Table 9.—Economic value of man, based on the premises that *F*, the functional ability, multiplied by *T*, the technical ability, multiplied by *C*, the competing ability, equals to *E*, the earning ability of the person, and that the gross economic value of man is the present value of all his earnings for a prospective working life, and that the net economic value of man is the present value of all his earnings less the present value of all his personal expenses for his prospective life.

The money values here given may be used either for the gross or for the net economic value of man, depending on whether \$1 per day is the gross or the net income per day for three hundred days of the year, namely \$300 per year, for a prospective working-life.

This table is computed on a 3½ per cent. discount basis. (¶ 6.)

At the Age of Birth	Money Value	Annual Increase	Percentage of Increase in 5 Years	Number Living at	No. Deaths in 5 Years	Percentage of Death in 5 Years
..	133.29	513
5	1,388.44	251.03	941.66	372	141	27.48
10	2,900.98	302.50	108.94	355	17	4.57
15	4,754.26	370.65	63.88	346	9	2.53
20	5,797.72	208.69	24.05	335	11	3.17
25	6,114.51	63.35	5.46	321	14	4.17
	Decrease	Decrease	Decrease			
30	5,985.71	25.76	2.10	307	14	4.36
35	5,664.43	64.25	5.36	291	16	5.21
40	5,262.30	80.43	7.13	275	16	5.49
45	4,784.29	95.62	9.08	257	18	6.54
50	4,178.96	121.04	10.57	237	20	7.78
55	3,420.67	151.68	18.14	215	22	9.28
60	2,413.00	201.52	29.45	189	26	12.09
65	1,141.72	254.25	52.68	156	33	17.46
70	19.10	224.73	98.33	118	38	24.35
75	609.32	125.68	3,293.50	79	39	33.05
80	-1,017.88	81.71	67.05	44	35	44.30

REPORT ON A SERIES OF FIFTEEN HUNDRED CASES OF ERRORS OF REFRACTION AND A BRIEF ANALYTICAL CONSIDERATION OF THE SYMPTOMS PRESENTED

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This paper which I have the honor to present before the Academy is a report on a series of refractions which I submit with the hope that I may be so fortunate as to either gain your approval or invoke such discussion that the end-result may be the same, a deeper research in the science of refraction and a further and more exhaustive study of the various phases of refraction which I shall discuss to-day.

This report is based on the case records of one thousand adults, private patients between the ages of 18 and 40, all of whom are of a class in which close work is more or less excessive. These adults were all examined under the complete cycloplegia of homatropin and cocain, their histories carefully recorded and final reports have been obtained from all.

Cases in which there existed muscular imbalances of such magnitude as to be productive of symptoms are excluded from the report and the findings submitted are composed of symptoms dependent on errors of refraction exclusively.

The second portion of this report comprises the findings in the examination of five hundred children between the ages of 5 and 16, who were examined under full atropin cycloplegia. As in the adults, only those cases are considered in which the difficulty is purely one of refractive error.

Your attention is first directed to summary sheet number one, which shows the comparative frequency with which the various errors were found in the series of one thousand adults.

SUMMARY SHEET NUMBER 1

Errors of Refraction in 1,000 Adults	Percentage
Compound hypermetropic astigmatism.....	34.1
Mixed astigmatism	23.6
Compound myopic astigmatism.....	23.5
Simple hypermetropia	8.9
Simple hypermetropic astigmatism	3.6
Simple myopia	3.6
Simple myopic astigmatism	2.4
Emmetropia	0.3

As you will note, in adults all forms of hypermetropia constitute 46.6 per cent. of all errors. Myopia in its simple and astigmatic forms aggregate 29.5 per cent. and mixed astigmatism is found in 23.6 per cent. of all the patients examined.

Let us now proceed on an analytical consideration of the various symptoms presented by these one thousand adults, taking each error separately. In the leading error of refraction found in adults, the following tabulation gives you at a glance the predominant symptoms and their frequency of occurrence.

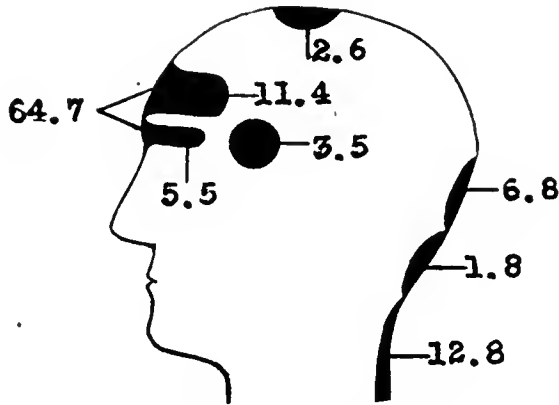


CHART NUMBER 1
Headaches—Compound hypermetropic astigmatism

OTHER SYMPTOMS MOST FREQUENTLY MENTIONED

Symptoms	Percentage
Rapid ocular fatigue.....	47.0
Tardy accommodation	9.3
Inadequate vision	11.4
Ocular pain	15.2
Palpebral irritation	34.7
Increased lacrimation	6.6
Photophobia	6.7
Nausea	6.1
Vertigo	3.2
Nervous irritability	22.3
Nervous depression	5.0

You will note that in compound hypermetropic astigmatism, the headache most frequently mentioned is the combined frontal and supra-orbital, 64.7 per cent. of all compound hypermetropic astigmats giving this form of headache as a leading symptom. In no other form of error of refraction is this headache present to such a degree, the nearest approach being in simple hyper-

metropic astigmatism, with a percentage of only 13.3. The next most commonly mentioned headache found in compound hypermetropic astigmatism is that in the nuchal region, the so-called "check-rein sensation." I consider the frequency and constancy of this symptom to be noteworthy. All other headaches total but 31.6 per cent., less than half the percentage of the combined frontal and supra-orbital.

Let us proceed to Chart Number 2. This presents for your consideration the tabulated symptoms found present in mixed astigmatism, which was found to be the existing error in 23.6 per cent. of all cases examined. The frequency of mixed astigmatism should be noted. Previous reports would indicate a much lower percentage and I feel that the increase in the percentage is due merely to our improved diagnostic technic.

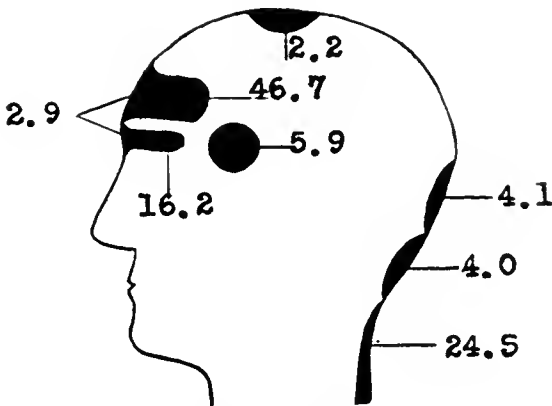


CHART NUMBER 2
Headaches—Mixed astigmatism

OTHER SYMPTOMS MOST FREQUENTLY MENTIONED

Symptoms	Percentage
Rapid ocular fatigue.....	50.3
Tardy accommodation	5.7
Inadequate vision	11.5
Ocular pain	16.1
Palpebral irritation	28.4
Increased lacrimation	10.8
Photophobia	12.7
Nausea	8.8
Vertigo	4.3
Nervous irritability	34.5
Nervous depression	7.2

In mixed astigmatism there is found the most frequent mention of the isolated supra-orbital headache. This is a comparatively infrequent symptom in the other forms of error of refraction. In mixed astigmatism the isolated supra-orbital pain was noted by 16.2 per cent. of these patients. The nuchal pain is present in 24.3 per cent., a higher percentage by 6.1 than is found in any other form of error. The presence of a well-defined supra-orbital headache in conjunction with nuchal discomfort or pain can well be called characteristic if not pathognomonic of mixed astigmatism. It is in this form of error that we find the greatest nervous manifestations present. More, however, will be said of that later.

In compound myopic astigmatism headache is found in the frequency as indicated on Chart number 3.

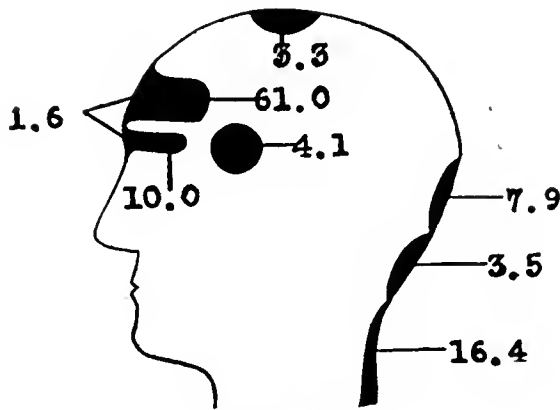


CHART NUMBER 3
Headaches—Compound myopic astigmatism

OTHER SYMPTOMS MOST FREQUENTLY MENTIONED

Symptoms	Percentage
Rapid ocular fatigue.....	40.3
Tardy accommodation	9.0
Inadequate vision	24.8
Ocular pain	13.4
Palpebral irritation	38.2
Increased lacrimation	8.4
Photophobia	10.7
Nausea	5.6
Vertigo	2.3
Nervous irritability	30.8
Nervous depression	8.5

In compound myopic astigmatism we find on analysis that the headache most frequently mentioned is the isolated frontal headache. Sixty-one per cent. of all headaches given as a leading symptom of this error is well defined in the frontal region. In marked contrast the combined frontal and supra-orbital headache is present in but 1.6 per cent. of the case histories, as against 64.7 per cent. found in the cases of compound hypermetropic astigmatism. Nuchal pain is mentioned by 16.4 per cent. of all these patients. All forms of headache other than frontal and nuchal constitute but 30.4 per cent.

The next chart is illustrative of simple hypermetropia, the least significant of all errors of refraction.

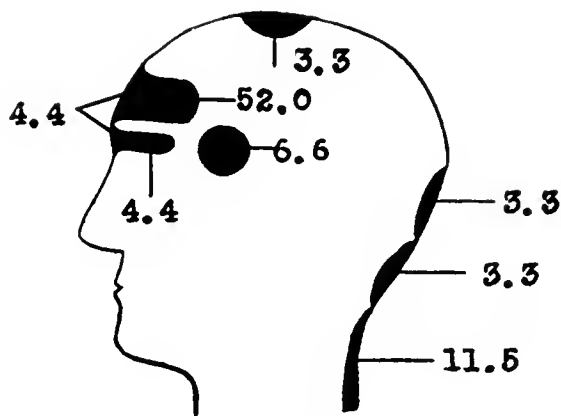


CHART NUMBER 4
Headaches—Simple hypermetropia

OTHER SYMPTOMS MOST FREQUENTLY MENTIONED

Symptoms	Percentage
Rapid ocular fatigue.....	44.5
Tardy accommodation	9.9
Inadequate vision	11.0
Ocular pain	16.7
Palpebral irritation	23.5
Increased lacrimation	4.4
Photophobia	13.3
Nausea	10.4
Vertigo	8.8
Nervous irritability	22.2
Nervous depression	0.0

As you note by Chart Number 4 in the cases of simple hypermetropia, we have a much more even distribution of all symptoms than in any of the foregoing charts. There is absent any characteristic preponderance of one form of headache as was found in the summarization of the other errors of refraction. Let us proceed to the consideration of the simple hypermetropic astigmatism.

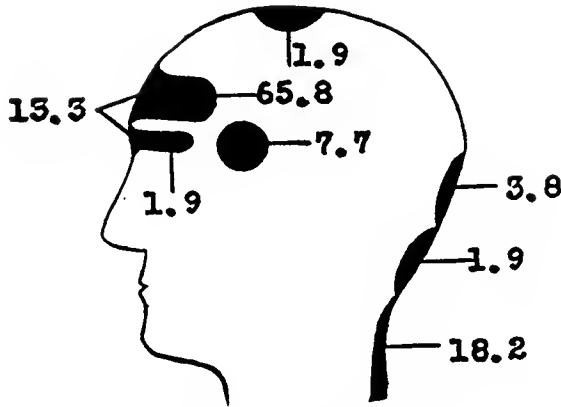


CHART NUMBER 5
Headaches—Simple hypermetropic astigmatism

OTHER SYMPTOMS MOST FREQUENTLY MENTIONED

Symptoms	Percentage
Rapid ocular fatigue.....	60.2
Tardy accommodation	12.4
Inadequate vision	3.8
Ocular pain	4.7
Palpebral irritation	46.7
Increased lacrimation	7.7
Photophobia	16.5
Nausea	9.6
Vertigo	7.7
Nervous irritability	35.0
Nervous depression	0.0

In simple hypermetropic astigmatism, as is shown above, the isolated frontal headache is represented by the highest percentage found in the entire tabulation and, as you see, 79.1 per cent. of all these cases mention pain in the frontal regions, that is in the isolated frontal and the frontal and supra-orbital in combination. This is a higher total percentage than is found else-

where. All other symptoms are fairly evenly distributed; but, as you see, we have a comparatively high percentage of nuchal pain.

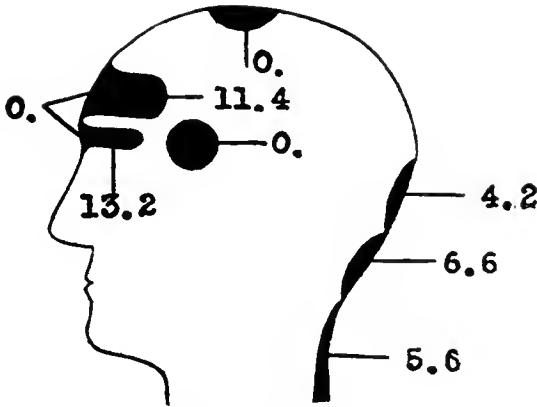


CHART NUMBER 6
Headaches—Simple myopia

OTHER SYMPTOMS MOST FREQUENTLY MENTIONED

Symptoms	Percentage
Rapid ocular fatigue.....	29.5
Tardy accommodation	15.7
Inadequate vision	74.3
Ocular pain	13.3
Palpebral irritation	25.2
Increased lacrimation	3.3
Photophobia	10.0
Nausea	6.7
Vertigo	0.0
Nervous irritability	0.0
Nervous depression	0.0

In simple myopia the comparative infrequency of headache is striking. Only about 25 per cent. of all myopes examined mention headaches at all and their headache was invariably described as being but a vague feeling of discomfort, usually supra-orbital or frontal in character, never a combined frontal and supra-orbital. It is well to note here that only in simple myopia and in simple myopic astigmia do we find no mention made of the combined frontal and supra-orbital headache. In this error alone is no mention made of any of the nervous manifestations.

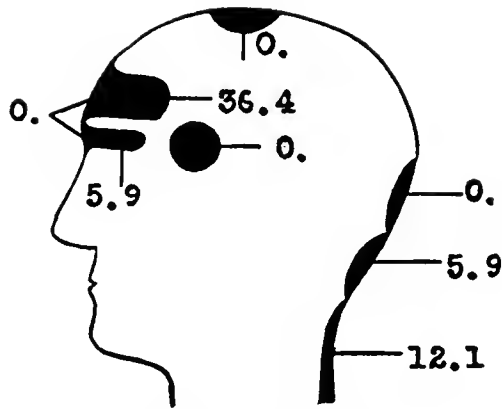


CHART NUMBER 7
Headaches—Simple myopic astigmatia

OTHER SYMPTOMS MOST FREQUENTLY MENTIONED

Symptoms	Percentage
Rapid ocular fatigue.....	26.4
Tardy accommodation	2.9
Inadequate vision	39.3
Ocular pain	0.0
Palpebral irritation	60.0
Increased lacrimation	18.2
Photophobia	12.0
Nausea	8.8
Vertigo	0.0
Nervous irritability	17.6
Nervous depression	0.0

SUMMARY SHEET NUMBER 2

Error of Refraction	Frontal with Supra-orbital	Frontal	Supra-orbital	Temporal	Occipital	Vertical	Suboccipital	Nuchal
Simple hypermetropia	4.4	52.0	4.4	6.6	3.3	3.3	8.3	11.5
Simple hypermetropic astigmatia	13.3	65.8	1.9	7.7	3.8	1.9	1.9	18.2
Compound hypermetropic astigmatia..	64.7	11.4	5.5	3.5	6.8	2.6	1.8	12.8
Simple myopia	0.0	11.4	13.2	0.0	4.2	0.0	6.6	5.6
Simple myopic astigmatia	0.0	36.4	5.9	0.0	0.0	0.0	5.9	12.1
Compound myopic astigmatia.....	1.6	61.0	10.0	4.1	7.9	3.3	3.5	16.4
Mixed astigmatia	2.9	46.7	16.2	5.9	4.1	2.2	4.0	24.3

In simple myopic astigmatism we note but slight difference from simple myopia except that there is a very slight increase in the frequency of headache. This and simple myopia are the only errors of refraction in which no mention is made of vertigo.

The foregoing charts have shown you the symptom classifications in the various forms of error of refraction. For the purpose of more rapid comparison, I have prepared summary sheet number 2, a tabulation of all forms of headache in all errors of refraction.

Further discussion of this summary sheet is unnecessary. One point which I wish to emphasize particularly is the constancy with which the nuchal pain, or check-rein sensation, is found present as a leading symptom of the errors of refraction. Even in simple myopia, which presents so few symptoms other than inadequate vision, we find this nuchal pain in 5.6 per cent. of the case histories. The highest percentage, 24.3, is found in mixed astigmatism. The extent of this nuchal pain varies from a slight sense of discomfort in the nape of the neck to a severe pain extending well down into the dorsal region. It is a significant fact that in every case complete relief from this pain came from the use of the correcting lenses. Its frequency of occurrence demands for it recognition as a prominent symptom of eye strain.

In order of frequency, I found the headaches in these one thousand patients to be located as follows:

1. Frontal
2. Nuchal
3. Frontal with Supra-orbital
4. Supra-orbital
5. Occipital
6. Temporal
7. Suboccipital
8. Vertical

I do not wish to give the impression that I am attempting to establish a symptom group index to errors of refraction, but it does impress me that this analysis has at least indicated that certain mechanical defects of the eye produce certain definite symptoms in a predominating percentage of cases and that further study along the same line is warranted.

We will now proceed to a brief consideration of the symptoms other than headaches presented by these patients. Summary sheet number 3 presents in condensed form the summarized tabulations.

In this tabulation of symptoms other than headache there are a few points of interest which are of significance.

Rapid ocular fatigue is a very constant symptom, being least mentioned in simple myopia and simple myopic astigmatism, the greatest frequency being in simple hypermetropic astigmatism.

Tardy accommodation is found in highest percentage in simple myopia and lowest in simple myopic astigmatia.

Inadequate vision is, as would be expected, highest in simple myopia, followed by the myopic astigmatia. In the hypermetropia and mixed astigmatia, it is of negligible frequency.

Ocular pain in the highest percentage is found in simple hypermetropia, followed by mixed astigmatia, compound hypermetropic astigmatia, compound myopic astigmatia, simple myopia and simple hypermetropic astigmatia, in the order named. Ocular pain is not mentioned in simple myopic astigmatia.

Palpebral irritation in 66.6 per cent. of all cases of simple myopic astigmatia is a prominent symptom. It is found in highest percentage in the various astigmatic forms of error.

Increased lacrimation, being largely dependent on lid irritation, is also found chiefly in astigmatic errors.

SUMMARY SHEET NUMBER 3

Error of Refraction	Rapid Ocular Fatigue	Tardy Accommodation	Inadequate Vision	Ocular Pain	Palpebral Irritation	Increased Lacrimation	Photophobia	Nausea	Vertigo	Nervous Irritability	Nervous Depression
Simple hypermetropia	44.5	9.9	11.0	16.7	23.5	4.4	13.3	10.4	8.8	22.2	0.0
Simple hypermetropic astigmatia..	60.2	12.4	3.8	4.7	46.7	7.7	16.5	9.6	7.7	35.0	0.0
Compound hypermetropic astigmatia	47.0	9.3	11.4	15.2	34.7	0.6	6.7	6.1	3.2	22.3	5.0
Simple myopia....	29.5	15.7	74.3	13.3	25.2	3.3	10.0	6.7	0.0	0.0	0.0
Simple myopic astigmatia	26.4	2.9	39.3	0.0	60.6	18.2	12.0	8.8	0.0	17.6	0.0
Compound myopic astigmatia	40.3	9.0	24.8	13.4	38.2	8.4	10.7	5.6	2.3	30.8	8.5
Mixed astigmatia....	50.3	5.7	11.5	16.1	28.4	10.7	12.7	8.8	4.3	34.5	7.2

Photophobia is found rather evenly distributed.

The nausea herein mentioned is, as you remember, not dependent on muscular imbalances and it is interesting to note its existence in all forms of error of refraction with relative constancy of percentage.

Vertigo also is fairly evenly distributed and constant throughout the series.

Nervous manifestations I have considered of sufficient importance to be charted separately, as is shown in summary sheet number 4.

This is a phase of eye strain symptomatology which is of greater importance than has generally been admitted. Gould was a pioneer in the exploitation of the nervous phenomena

resulting from eye strain, and although his extreme deductions were not generally accepted, it cannot be denied that he established as truths what many had denounced as impossibilities. I think it is generally accepted now that nervous manifestations are among the most frequent and distressing of symptoms arising from eye strain.

SUMMARY SHEET NUMBER 4

	Simple Hypermetropia	Simple Hypermetropic Astigmatism	Compound Hypermetropic Astigmatism	Simple Myopia	Simple Myopic Astigmatism	Compound Myopic Astigmatism	Mixed Astigmatism
Nervous Irritability.....	22.2	35.0	22.3	0.0	17.6	30.8	34.5
Nervous Depression.....	0.0	0.0	5.0	0.0	0.0	8.5	7.2

In this tabulation I wish, particularly, to call your attention to the absence of the depression type of nervous manifestation in all but the compound and mixed astigmias and to the total absence of nervous irritability and depression in simple myopia. Seventy-five per cent. of the patients whose nervous manifestations are here recorded have presented final reports of complete recovery from the nervous manifestations. The remaining 25 per cent. are negative or only faintly positive. I have observed that the lower forms of astigmatic error are more prolific of nervous manifestations than are the gross errors of refraction. The gross errors result in such great reduction of visual acuity that nature very quickly realizes the futility of any attempt at correction. I am in accord with the contention that in those cases in which we find one eye with good visual acuity and the fellow-eye with a gross error of refraction, the disuse of the weaker eye is frequently but a protective measure against the establishment of nervous manifestations, the inevitable sequel of ocular strain.

The next chart, summary sheet number 5, is a graphic presentation of the various errors as found in the series of five hundred children in comparison with the findings in the series of adults.

From this summary sheet very interesting deductions can be made. Your attention is first directed to column 1 which shows the percent. of normal eyes found in adults to be 0.3 per cent., or three out of the thousand cases reported. In chil-

dren the percentage is 19.4, representing 97 children out of 500 examined, or 194 out of every thousand, as against 3 out of every thousand adults. In the simpler forms of error of refraction, simple hypermetropic, column 2, simple hypermetropic astigmatism, column 3, and simple myopia, column 5, we find present a relatively high percentage in children and a low one in adults.

SUMMARY SHEET NUMBER FIVE

		%	
1. EMMETROPIA		0.5	ADULTS
		19.4	CHILDREN
2. SIMPLE HYPERMETROPIA		8.9	ADULTS
		27.9	CHILDREN
3. SIMPLE HYPERMETROPIC ASTIGMIA		3.6	ADULTS
		14.0	CHILDREN
4. COMPOUND HYPERMETROPIC ASTIGMIA		34.7	ADULTS
		21.6	CHILDREN
5. SIMPLE MYOPIA		3.6	ADULTS
		5.4	CHILDREN
6. SIMPLE MYOPIC ASTIGMIA		2.4	ADULTS
		2.1	CHILDREN
7. COMPOUND MYOPIC ASTIGMIA		25.5	ADULTS
		1.6	CHILDREN
8. MIXED ASTIGMIA		25.6	ADULTS
		4.8	CHILDREN

When we consider the more complicated forms of error, compound hypermetropic astigmatism, column 4, compound myopic astigmatism, column 7, and mixed astigmatism, column 8, we observe that these conditions are found present in a high percentage in adults and in a low one in children.

It is self-evident that the normal eyes and those with the simpler errors assume in later life the more complicated forms of refractive error. You will note that the increased percentage

in the first, second, third and fifth columns is equalled by the loss of percentage in the fourth, sixth, seventh and eighth columns. In this computation there is found an error of 0.1 per cent. What excuse is there for allowing these ninety-seven children with normal eyes to become the victims of eye strain with its attendant handicap? There is no excuse. This transition from normal to abnormal is not a natural process nor is it unpreventable.

Within one generation this chart would be greatly modified if we were but able to give these children's eyes a fair chance. It is a reproach on us as ophthalmologists that these simpler forms of error are allowed to develop into the complicated astigmatic forms. Our educational system in this country is largely at fault. No child should be permitted to enter the public schools until he is 8 years of age. My observation in the study of errors of refraction in children leads me to believe that great harm is done during the sixth and seventh years, the first two years in school. Medical inspection of schoolchildren has accomplished vast good. In addition to that, there should be instituted in each city a corps of ophthalmologists to inspect the eyes of all children. Not a casual test-chart examination, for I claim that normal visual acuity in a child means nothing, but there should be a thorough consideration of each child's case, made possible by careful observations recorded on special blanks by the child's teacher. A record of a child's deportment, mental, physical and nervous, is of far more diagnostic value than the usual test-card measurement.

The children whose histories comprise this report were under very careful observation and on specially printed observation sheets a record of all symptoms was noted. The tabulated symptom groupings of these five hundred children are practically negative, so far as the usual eye strain symptoms are concerned. Headache of a definite type was found so infrequently as almost to be conspicuous by its absence. There is a corresponding paucity of all the other usual symptoms and the observation of subjective and objective symptoms was of such irregularity and vagueness as to render classification impossible. There is one exception to this negative report and that relates to the nervous manifestations observed. Over 70 per cent. of these children presented either nervous irritability or nervous depression and in 60 per cent. of these cases complete relief came with the elimination of the eye strain. Many of these children suf-

ferred from what Dean Emerson of the Indiana University School of Medicine has described as the "painless headache," the psychic depression which is the equivalent of the headache. This psychic depression type is very frequently observed and the following case history is illustrative of a great many cases which have come under my observation. I cite this not as an unusual case, but as a very common one.

R. S., Feb. 8, 1913. Aged 10 years. Nearly every afternoon Richard comes home from school and after putting away his books he throws himself down on the davenport and cries. He will cry for from a few minutes to nearly two hours. He never complains of headache, his eyes do not hurt and he cannot give any reason for crying. The child is not supersensitive, his feelings are not easily hurt, his work in school, although poor, is not a cause of worry or mortification and he gives no cause for crying, merely saying that he can't help it. This crying is not of the hysterical type in any sense of the word. No source of physical irritation is present. He rests well at night, but frequently has bad dreams, the same dream often recurring on successive nights. With the exception of nervous irritability and inability to concentrate there were no symptoms available. On examination the vision in each eye was found to be 20/20. He was examined under atropin cycloplegia of four days' duration. Both fundi normal. The skiascopic examination revealed a low grade mixed astigmatism for which the full skiascopic findings were prescribed. The boy's recovery was rapid and uneventful and at the present time, one year and eight months since the day of examination, there has been no return of symptoms.

Psychic irritation resulting from eye strain is a very common thing in the observation of all of us. This psychic irritation ranges in severity from a mild irritability to a form choreic in character or in the extreme cases to a degree identical in onset and symptoms to an epileptic seizure. To relate these case histories would be but repetition of what you yourselves have observed.

Other than these observations, I admit failure to establish any facts of importance. To establish scientific data of value means the careful study of thousands of cases instead of hundreds. However, the study of these five hundred cases has convinced me of two things, the constancy of nervous manifestations and the inconstancy of all other symptoms generally recognized as being ocular in origin.

Before concluding this paper I wish to enter into some detail on the method of examination, mentioning only the departures from the usually accepted technic.

CYCLOPLEGICS EMPLOYED

Solutions of atropin sulphate, 0.5 per cent., are used with children between the ages of 9 and 15. For younger children the strength is proportionately lower. The process of atropinization extends over a period of four days.

HOMATROPIN AND COCAIN

I find this combination dangerous and inefficient in children. In adults I am convinced that a disk containing $\frac{1}{50}$ of a grain of homatropin and cocain is sufficient, in the vast majority of cases, to produce complete cycloplegia in a period of time varying from twenty to sixty minutes. In less than 5 per cent. of my recorded cases has it been necessary to increase this dosage. My method of determining the onset and progress of cycloplegia is that advocated by Dr. Lucien Howe in his work on "The Muscles of the Eye," (p. 157, vol. i). The observations of Straub, as quoted by Dr. Howe, confirm my findings in regard to the dosage required to obtain full and complete cycloplegia. My observations are at variance with Straub's in that I have secured complete relaxation of the muscles of accommodation in less time. This, in some measure, may be accounted for by the fact that Straub used homatropin alone, while in my experiments I employed it in conjunction with cocain hydrochlorid. The age of the patients doubtless influences the results. Among many of my confrères it is the custom to use a 2 per cent. solution of homatropin and cocain, one drop in each eye at intervals of ten minutes, extending over the period of an hour. I believe that used in that manner there is a useless waste of time and homatropin, as well as an unnecessary amount of discomfort to the patient, resulting from the prolonged cycloplegia and mydriasis. My contention probably will meet with opposition, but I believe that a series of carefully conducted observations will convince the most skeptical.

MYOTICS

My routine practice is to employ physostigmin salicylate to counteract the effect of the homatropin and cocain. I can commend its use to you as being a great convenience to your patients, in that in proper dosage it restores the accommodative function in from ten to sixty minutes. From the standpoint of safety it constitutes a valuable precautionary measure. Three-fourths of the patients recover their ciliary activity in an average of

thirty minutes following the instillation of the physostigmin. I have used it as a routine practice for five years and thus far have observed no untoward effects in any case. Its use in young adults is not advisable, as it frequently causes an intense ciliary cramp with its attendant severe headache.

METHOD OF DETERMINING ERRORS OF REFRACTION

When I first essayed the determination of errors of refraction I depended to some extent on my skiascopic measurements, modifying them, however, by the subsequent trial-case examination. If the two methods were at variance I would abide by the lenses accepted at the trial case by the patient. Subsequent and final reports from a large percentage of my patients were unsatisfactory. In the course of time I was brought to the full realization of the fact that there is nothing more untrustworthy than your patient's visual judgment. With improvement and development of the technic of skiascopy I gradually became less dependent on the subjective tests. To-day I feel that skiascopy offers the only scientific and exact method for the determination of errors of refraction. The test-letter, trial-case method I believe to be a dangerous, unreliable method and to substitute for your skiascopic findings a lens selected by your patient is but the casting aside of scientific accuracy, under your own control, for the unintelligent guesswork of your patient. Refraction without recourse to any subjective tests is to my mind the ideal method of determining errors of refraction and the final reports which I have from patients examined and prescribed for by this method during the past seven years have convinced me of the absolute reliability of the objective determination of errors of refraction. In order to obtain results, the most minute details of the science must be observed absolutely. Unfortunately most of our text-books lead us to the conclusion that skiascopy can be mastered in a short time. An article in one of the medical journals states, "Refraction is easy to learn." I claim that scientific skiascopy demands a most thorough training and a more exacting technic than any of the specialties and that the mastery of the intricacies of the art remains yet to be accomplished. My claims for this method are made in no conceit, for I possess no originality of method or appliances, but I do claim that anyone who will develop the technic of skiascopy will be rewarded by having at his disposal a method of correcting errors of refraction which will give him better results than by the subjective tests.

Further discussion would be a digression from the subject at hand, but I do hope that what I have said may possibly stimulate interest in this most important branch of ophthalmology and that scientific skiascopy may assume the importance which it deserves.

In closing I beg to call your attention once more to the summarized symptom groupings found present in adults and to impress on you the constancy and frequency of the various indications of ocular strain; also to emphasize the importance of recognizing nervous manifestations as being the most constant symptoms of eye strain in children; and last, to ask your consideration of what I am convinced is fact, that the unscientific uncertainty of subjective tests must and will give way to the scientific precision of the objective method of determining errors of refraction.

ROUTINE REFRACTION PROBLEMS

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SYNOPSIS

1. Completeness or incompleteness of cycloplegia. Diagnosis and significance of latter.
2. Therapeutic uses of cycloplegia barring its employment in determining static refraction.
3. Nature's compensative efforts.
4. Prism exercise.

The routine refraction problems to which I ask attention are:

1. Uncertainty regarding completeness of cycloplegia and its meaning.
2. Therapeutic uses of cycloplegics, other than that for uncovering latent error.
3. Interpretation of muscular imbalance, with special reference to the bearing on correction of ametropia, treatment of certain forms of heterophoria at the reading distance, nature's ability to counteract imbalance, and prism exercise.

First, a word regarding the necessity of a cycloplegic examination. It gives, or is intended to give, and should give static refraction—exact refractive power of the eye without accommodation. That the eyes are never used without accommodation, is no argument against the necessity of our knowing how much excessive ciliary activity enters into distant vision, or how much is suppressed in near vision. The knowledge of these activities is an essential part of successful refraction work. Nothing gives this save complete cycloplegia.

Most of us like to use homatropin. Probably we begin with it unless evident presence of a high error or a visible squint contra-indicates it. One other condition always decides me against homatropin—apparent need of concave glasses in young persons, 25 or younger. As to the amount of homatropin needed, I am inclined to think that five or six applications of a 2 per cent. solution, especially if preceded by the cocain and homatropin disks containing $\frac{1}{50}$ grain of each, will give all the results obtainable by more prolonged use. There are exceptions, but I have rarely found any change after this amount of use.

Is there any way of determining whether or not we have secured cycloplegia? I have had a few such experiences as the following:

CASE 1.—A young girl showed under homatropin a $\frac{1}{2}$ D. of + astigmatism with 1 of hyperopia. Atropia doubled the hyperopia and its use for three days did not alter this amount, which was accepted as the total error. Yet this child came back after six months' wear of + 1.50 with a manifest hyperopia of 3.50 D. One and a half D. hyperopia had remained latent in spite of prolonged use of atropia, but had gradually become manifest behind glasses.

CASE 2.—A girl of twenty, with myopic refraction, showed 4 D. myopia after a week's use of atropia. The error was confirmed by shadow-test. Full correction was ordered with normal vision, orthophoria at 6 meters, exophoria of 3 degrees at 33 cm. One month later myopia *with* accommodation was only 3 D. and she had an internal squint behind her glasses. Correction of the cycloplegic myopia had produced one D. of hyperopia and defective relative accommodation had produced squint.

I do not know how to avoid such errors, which, fortunately, are not frequent. Really, we have no absolute guarantee that the ciliary muscle is completely paralyzed. When dealing with other than myopic refraction, retention of the best distant vision through a concave glass of $\frac{1}{2}$ or $\frac{3}{4}$ diopter throws doubt on completeness. But, after all, the best available tests are consistency of subjective findings with objective measurements, and permanency. By permanency I mean no variation in the axis of the astigmatism and no changes from moment to moment in the spherical or cylindrical error. An axis may vary within 10 or 15 degrees; an error may appear spherical, only after a moment to seem astigmatic. The inference is either asymmetrical refraction, of which the dilated pupil is the only evidence, or incomplete paralysis. Usually the sequel shows that the latter inference is justified at the outset. But why should the ciliary muscle be so rebellious? What keeps up the irritation? You all recognize the class of patients and know how bothersome they are.

Some years ago I adopted the following as a routine primary examination with every refraction patient: history, ophthalmometric measurements, ophthalmoscopic examination, shadow-test with undilated pupil, manifest error, muscular balance for distance and near, and accommodative range for what we used to call diamond print, but which is now marked "0.5 D." Young persons, 25 or under, should have a range from 10 or 15 to 50

cm. In a certain number I found a shortening of the far point and a recession of the near. Then two other things appeared. These were the very patients who showed resistance to cycloplegics, and after I had, or thought I had, corrected the ametropia, the accommodative anomaly relapsed. Usually there was an increase in refraction for distance, but always this peculiar form of ciliary spasm. Renewed cycloplegic tests confirmed former findings. Evidently something was at work on accommodation. What was it? One of the early cases (my own daughter at the age of 11) showed after a few months of this variation a spot of choroidal exudate too close to the fovea for comfort; but it cleared up. Later she developed what the surgeons called chronic appendicitis, underwent operation, and since then—ten years—has been comfortable with her spherical correction. The query arose, what is behind chronic appendicitis in the way of metabolism? What are the remote effects of chronic adhesive appendicitis? I believe there is a good deal here our surgical friends might look into with advantage. Since then I have seen numerous cases presenting the accommodative condition described, with stubborn resistance to cycloplegics, and systemic examinations have shown excessive indicanuria and other evidences of intestinal disorders. I have mentioned in another paper the case of a girl who had this relapsing ciliary spasm for two years and then developed interstitial keratitis. My own observations during these years had been too narrow. I do not care to lengthen this paper with cases, but I am convinced that many systemic causes of uveal inflammation and other irritants whose action we cannot explain may produce uveal irritation before something happens big enough to call attention to the underlying cause; that a not uncommon manifestation of the irritation is some ciliary anomaly; that the presence of either of the two already discussed—resistance to cycloplegics and recurrent ciliary spasm—calls for systemic study.

The use of cycloplegics in restoring normal eye function, apart from uncovering static error, was first brought home to me by my friend Dr. Risley, to whom all of us owe so much for making us think. The classes of cases to which he called my attention were our failure to relieve symptoms in spite of work the correctness of which we were sure, and the significance of blurring of the neuroretinal margin in uncorrected ametropia. In the first class symptoms continue. We use a cycloplegic a second or third time to verify our first results. No reason to

alter glasses is found. Or another to whom the patient goes does the same, and later we learn results. Relief is finally obtained, usually without change in the original glasses. The curative agent has been the repeated rests afforded by ciliary paralysis and lessening of hyperemia. The results of eye-strain were too pronounced to disappear at once. In the second class the neuroretinal blur is the result of uveal hyperemia. It shows itself at this particular place because here only the uveal and retinal circulations meet. I think the nerve head is nearly always of a reddish hue, deeper than normal. While this lasts, the eyes are irritable. Cycloplegia is sometimes needed for weeks before normal appearances are observed, and rarely does comfort come before they are restored. I have observed two classes of patients who are greatly helped by occasional abeyance of accommodation. One has been already discussed—those who show repeated ciliary spasm in spite of refraction correction. The underlying cause may not be discoverable, or, as in diabetes, for instance, is incurable. Eye irritation continues. Such patients are greatly aided by occasional cycloplegia. They soon learn by increasing discomfort when they need it. My habit is to let them use homatropin Saturday night. Then they are ready for work Monday morning. I have patients who go through this process about once in six weeks. The other class is made up of those whose environment compels them to push the eyes whether or not they are able to work. Book-keepers, teachers, and stenographers are types. Eyes, like other parts of the body, have working limitations. If circumstances drive the owner beyond these limits he or she suffers. Homatropin gives the eyes a temporary rest and a fresh start.

To a greater or less degree every clinician is a law unto himself in the study of heterophoria. Tests with the phorometer, Maddox rod and kindred appliances are thought to give the tendency of divergence when eyes accustomed to binocular vision cannot obtain it. Such tests do not of themselves tell whether the imbalance is primary, i. e., due to excessive or defective strength in one muscle, or secondary, i. e., the result of abnormal innervation, usually due to ametropia. They indicate in only an incomplete degree the need of the muscle itself for any treatment other than correction of ametropia. Decision on this point must be withheld until other tests are used, and often until time reveals. In a general way, the balance test indicates certain things as advisable in refraction correction, and

experience shows these to be reliable. We aim to force latent hyperopia into manifest, as nearly as possible to total correction when the balance is esophoric; we anticipate modifying this to a greater or less degree when the balance is exophoric. We give a total correction to myopia, provided the fundus is good and acuity normal, and aim the more strenuously to this end if the balance is exophoric. We anticipate trouble if it is the other way.

With these general principles, it will probably be best if I try to set forth how I study heterophoria in routine work—not because I am sure my method is the best, but because it has seemed to stand me in good stead, and in so far as it is wrong, I should like it corrected. With the patient wearing from the trial-case glasses which correct the total error as nearly as is consistent with the best visual acuity, provided this is not excessively low from intra-ocular lesions or amblyopia, muscular balance is taken at 6 meters and at 33 cm. I regard the normal balance at these distances as orthophoria and exophoria of two to five degrees. If some form of imbalance be present, there should still be this relation of a low near exophoria as compared with the distance balance. For instance, an esophoria of 4 degrees would demand, in the near, orthophoria or 1 or 2 degrees of esophoria. I use the term “normal” because this relative balance between distant and near vision is that which obtains in comfortable eyes. But balance tests are not the only means of examination. We have the various “ductions,” vertical and horizontal. What is their value? It seems to me that this can be best estimated if we bear in mind the relations existing between the muscles themselves when called into action. When we test for what we call adduction at 6 meters, what are we looking for? The normal combined action of the interni is in connection with accommodation. Visual clearness is the guiding sensation in all visual acts. Hence, effort to maintain single vision at 6 meters, through prisms, bases out, will be resigned when vision becomes blurred through innervation to the ciliary muscle, produced by increasing efforts to innervate the interni. When the patient learns how to separate accommodation from convergence, as he soon does by one or another of the various forms of prism exercise (Gould’s, Duane’s, or Savage’s rhythmic exercise), adduction is doubled, tripled or quadrupled in a short time, sometimes in a few minutes. Is this a test of intrinsic muscular power of the interni? Have we really

increased real muscular power to such an extent in a few minutes? I do not think either supposition is true. We have simply trained the interni to act independently of the ciliary muscle—developed what Donders called positive relative convergence. That defective relative convergence can be a real cause of asthenopia, there is no doubt. I shall speak of this in a moment; but its cure is not due to actual increase of muscular force. There seems to be a minimum of independent action of the interni, below which comfort cannot exist. This independence is what we call adduction, and its normal minimum is generally accepted as about three times the power of the externi, or abduction. Take, again, the vertical muscles. They have no absolutely independent action. The superior rectus acts with the inferior oblique, and the inferior with the superior oblique. When we say that infraduction, for instance, is 1 degree, do we know how much has come off the accepted normal of about 3 degrees by torsion or lack of it in the obliques? I doubt it. I have seen patients struggle with a 2-degree prism, turn the head a little and at once fuse vertical displacement. The external rectus is the only extrinsic muscle which has its own separate nerve supply, and no entangling alliance. I may be wrong, but for a long time I have relied on this separate nerve supply as the key to the real balance between the lateral muscles. Years ago the late Dr. Noyes' book gave 5 to 8 degrees as normal abduction. Less than this suggests real externi weakness, more, abnormal strength, and consequently excessive burden on the interni.

To summarize as a basis for clinical study; it seems to me (1) we should regard the combined action of the vertical and obliques as one action, not because it would not be desirable to separate our estimation of the work of each set, but because we do not know how to do it. The normal prism power of this combined action is about 3 degrees. (2) We should regard adduction as nothing more than the power of the interni to act at a fixed distance, independent of the ciliary muscles. (3) Abduction gives an index to the real muscular power of the externi.

With this as a working basis, I want to call attention, briefly, to two safeguards, as I term them, in estimating the significance of heterophoria in general, and to the meaning of certain peculiar departures from normal balance. If my own experience is reliable, any form of heterophoria, and to almost any degree, may be demonstrable by the phorometer, and yet the eyes give

no trouble. Nature seems to possess almost unlimited powers to regulate innervation in the interests of comfort, despite what a balance test may show. A balance esophoria may be rendered harmless by lowered interni, or high externi innervation. I saw recently a school teacher, comfortable with a spherical correction of $+1.5$ sp. yet with a phorometer esophoria of 10 degrees. In spite of this, abduction was 12 degrees. Why a balance test indicates weakness, and prism test, excessive strength, in a muscle with its own nerve supply, is a little hard to understand. A fraction of this esophoria would torture another patient.

The two tests I have found useful in distinguishing harmless from important heterophoria, and, at the same time, indicating whether or not prism help is needed and its amount, are the parallax test of Duane, and the red-glass test of Savage. Working independently, Verhoeff developed a test identical with Duane's and called it the "shutter" test. The prism, base toward the weak muscle, which stops the parallax movement, or prevents diplopia by the red glass, serves as an index of prism correction. Usually it is not over one-half or one-third of phorometer imbalance, and according to its amount and associated ametropia, indicates no prism or its strength.

There are two peculiar forms of imbalance at the reading distance to which I want to make brief allusion. One has been presented from time to time by Theobald under the caption, Subnormal accommodation in young persons. He believes that this condition, refraction errors having been corrected, is indicated by an esophoric balance at the near as compared with distance. For instance, orthophoria at 6 meters with esophoria at 33 cm. would indicate excessive innervation to the interni, in turn produced by excessive ciliary innervation. This, he argues, is needed because of intrinsic asthenia of the accommodative muscle. Convex lenses restore the normal near exophoria, and the lens doing so should be added to the distance correction for near work. Many troublesome cases can be relieved in this way, but the doctrine can be carried too far. The error, if it be one under such conditions, is often found in myopia; yet, a short time's wear of total correction develops the unused accommodation and restores the near exophoria which both he and I believe normal. The condition is also found in uncorrected astigmatism and rights itself after correction, for reasons which are entirely apparent. It will also clear up under correction of simple hyperopia. But the meaning (ciliary asthenia) to be attached to this

near imbalance, is, I think, indisputable. The sole question is whether the asthenia is essential or symptomatic. The accompanying refraction error or a little time will tell.

The other form of near imbalance is an excessively high near exophoria; for instance, orthophoria in the distance and 12 to 15 degrees exophoria in the near. In my experience it is usually seen in the hypermetropia of adults. It is a very troublesome cause of asthenopia, and is not cured by wearing prisms, base in. I am under the impression that the following hypothetical explanation was given by B. Alexander Randall in a discussion at the American Ophthalmological Society. It is an overdoing of separation of accommodation from convergence. Positive relative accommodation or convergence—to preserve Donders' term—has wide limits and is capable of easy development. Uncorrected hyperopia forces excessive ciliary stimulation. This necessitates relative suppression of convergence to preserve visual lines. The individual overdoes it, i. e., suppresses convergence too much. I think the treatment indicated is prism exercise, provided correction of ametropia alone does not afford relief. Sometimes it does, usually it does not, for the spherical glass lessens stimulation and the muscular fault is increased. This form of heterophoria is the only one I have ever cured by prism exercise. Patiently and repeatedly I have tried to develop the vertical and external recti, with no results. I have had only occasional improvement with distant exophoria associated with high abduction; this, I take it, is better treated with prisms, base in. It is in ridding one of a vicious habit, formed by the necessity of seeing in spite of hypermetropic strain, that prism exercise gives best results.

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NEW LIGHT ON THE THEORY OF ACCOMMODATION, WITH PRACTICAL APPLICATIONS

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Kepler, who was the first to understand how light is refracted by the eye and images are formed on the retina, perceived the necessity for a means of changing the focus to adjust the eye to different distances—accommodation as we call it. Many guesses were made in the attempt to explain how this is accomplished, and a voluminous literature grew up around the subject of accommodation.

Some thought the eye was exempt from the laws of ordinary lenses and could manage to form clear images of objects at different distances without the need of changes in its refracting apparatus. Some found in the contraction of the pupil, which occurs when one looks at near objects, a sufficient explanation of the power of the eye to accommodate. Others advocated the theory that the cornea changed its curvature and even thought they detected changes by as careful measurements as they knew how to make. Another explanation was that the lens was movable and by shifting its position nearer the cornea made accommodation for near objects possible. Measurements showed that, as a matter of fact, the anterior pole of the lens does move forward in accommodation. Still another group of able men thought to explain the riddle by the theory that the eyeball itself became longer, under the influence of the external ocular muscles. Scheiner, Descartes, John Hunter, Thomas Young, Cramer, Purkinje, Graefe and finally Helmholtz advocated and indeed proved that accommodation takes place by a change in the shape of the crystalline lens.

Copious discussions and experiments were made to show how this change took place and exactly what the change was. Helmholtz, Tscherning and Hess are the principal names in these controversies. The overwhelming majority of ophthalmologists to-day follow Helmholtz and Hess in the belief that the ciliary muscle, by its variously arranged fibers (not to be too diagrammatically thought of as one group radial and one group circular) acts as a relaxer of the suspensory fibers of the zonule which supports the lens. It does this both by pulling forward the

posterior ends of these fibers and by decreasing the circumference of the ring which this ligament forms. The relaxation may go to the point of permitting the lens to become slightly movable, so that it falls, under the influence of gravity, toward that side of the eye which is lowest, depending on the position of the head. Under the influence of this relaxation the pupillary area of the lens becomes more sharply curved and so of shorter focal length, while the periphery of the lens, at least in many eyes, becomes more flattened; but by the contraction of the pupil that part is excluded from participating in the formation of images under ordinary favorable conditions.

When the suspensory ligament is relaxed, the lens, due to its inherent elasticity, including that of its capsule, takes its proper form. Hence, as Hess has pointed out repeatedly, the ciliary muscle does not produce changes in the lens in proportion to the strength of its contraction. All the ciliary muscle can do is to relax the zonule, the lens itself must change its own shape, not by virtue of any force applied from the outside but by its own inherent forces. The amount of contraction of the ciliary muscle necessary to fully relax the zonule depends therefore on the capacity of the lens to take up the slack by changing its form and so diminishes as age advances. Any surplus contraction simply makes the zonule slacker and so permits the lens as a whole to wobble or to sag toward the lowest point.

Owing to the important share taken by the lens itself, it must be duly and carefully considered in studying any of the problems of accommodation, such as fatigue of accommodation, the time element in accommodation, the action of drugs in accommodation, etc. Landolt, basing his statement on clinical experience, has said that for continuous near work one can use two-thirds to three-quarters of his total range of accommodation. If, for example, he has 6 D. of accommodation and 1 D. of hyperopia, he can work continuously at 33 cm., because he is using 1 D. to correct his hyperopia and 3 D. for the focusing at 33 cm., making 4 D. in all; this is two-thirds of his total of 6 D. He can do this with comfort. This is a much larger proportion of the total power of a muscle than can be used in the case of the external ocular muscles or of the ordinary skeletal muscles and requires some explanation.

This explanation is to be found partly in the fact that when we measure the range of accommodation, say 6 D., we are not measuring the total strength of the ciliary muscles, since this

would probably suffice to relax the zonule enough to produce 12 D. or more of accommodation if the lens could take up the slack. This hypothetical maximum range is called by Hess the total range and the corresponding near point the latent near point, while the ordinary or actual range he calls the manifest range. Thus a man of 33 who has 6 D. of manifest accommodation and uses 4 D. for continuous work is not using two-thirds of the total power of his ciliary muscle; since, if his lens were of such consistence and elasticity as to be able to take up all the slack, he could probably accommodate more than double the 6 D., and so is using less than one-third of his total muscular power when he is working with 4 D. of accommodation.

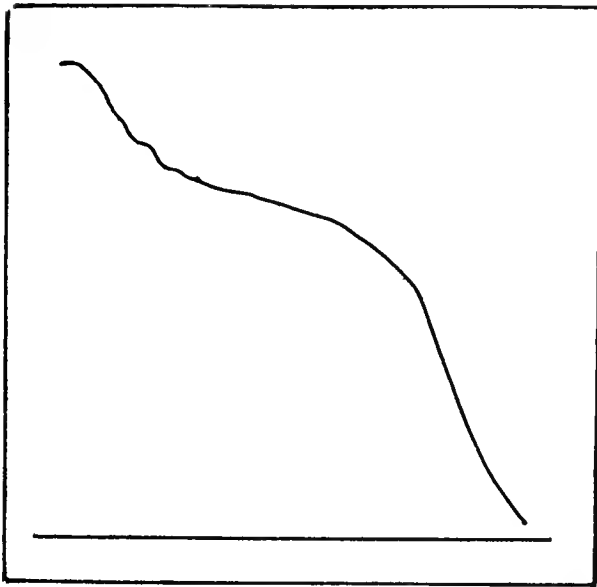
A second reason why we can use a larger percentage of the total effective power of the ciliary muscle is that it is a smooth muscle, more like the sphincter muscle of the bladder and other hollow viscera than like the striped skeletal muscles. It is characteristic of these smooth muscles that they maintain a constant considerable tonus or contraction without any sensation of fatigue. The circular fibers of the iris constitute the sphincter muscle of the pupil; a moderately strong contraction of this can be kept up indefinitely, only when the contraction is extremely strong is it painful or fatiguing. Similarly, the ciliary muscle, when required to adjust the eye for an ordinary reading distance in a patient who is not presbyopic or otherwise abnormal, can do this and keep it up indefinitely without discomfort. Only when the contraction is extremely strong is it painful or fatiguing and then far less than an equally strong contraction in a striped muscle would be. Some of the older writers called the ciliary muscle the sphincter muscle of the lens.

Wishing to make some tests of fatigue of accommodation, that is of the ciliary muscle and nerve, the following experiment was one of those tried. As will be seen, the result was quite different from what was anticipated.

The punctum proximum and punctum remotum were taken of the right eye of each subject and he was required to fix and focus a small test object or read fine print kept at his near point for a period of time which varied in different cases from less than a minute up to more than an hour, the punctum proximum being taken at short intervals and both punctum proximum and punctum remotum taken at the end. It was thought likely that as the ciliary muscle became fatigued the punctum proximum would recede. We were uncertain how the punctum

remotum would behave. As a matter of fact the punctum remotum is brought nearer, and this shows itself by the patient requiring a minus spherical lens to be added to his distance correction (previously ascertained) in order that he may now obtain his maximum visual acuity at 6 meters. If the eyes are kept shut or otherwise rested, this gradually passes off. The recovery takes usually ten minutes or more.

A greater surprise was the behavior of the punctum proximum. So far from the near point receding, it is brought nearer than at the beginning. Even after an hour's reading at the near



FATIGUE CURVE

Fig. 1.—Middle finger lifting 3 kg. every two seconds. Curve shows diminishing height to which weight is lifted = fatigue (after Maggiora).

point, during which time the accommodation is constantly maintained at the maximum degree, the subject can see clearly at a nearer point than at the start. Moreover, the subjective sensations of fatigue are surprisingly small. This is often remarked by the subject of the experiment. Indeed, when we began we did not think that we could maintain maximum accommodation for so long a time. The well-known investigations of fatigue of striped muscles, e. g., of the finger with the ergograph or of the gastrocnemius of the frog with the myograph, show that maximum contractions can be kept up for only a short time

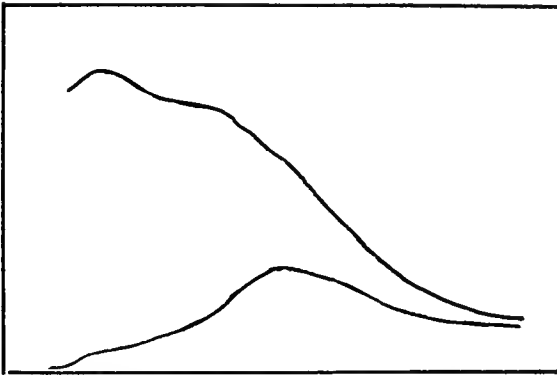
before their strength diminishes (Figs. 1 and 2). It was therefore an unexpected result to find that even after an hour the curve of accommodation did not, in all cases, show clear evidence of having passed the maximum and begun to fall off. On the contrary the curve, in some cases, still showed increase, but at a much slower rate. In other cases the curves showed signs of falling off after about half an hour.

Now, the explanation of the change in the range of accommodation whereby the punctum remotum is brought temporarily nearer and the punctum proximum is also brought temporarily nearer is a twofold one. In the first place the ciliary muscle is, as stated above, a plain or smooth-fibered muscle and partakes to some extent of the peculiarities of that type of muscle. Accordingly, it is not surprising that it shows a tendency to contracture—what we speak of in ordinary clinical work as “spasm of the accommodation.” We must discriminate between tone and contracture.

Howell's Text-Book of Physiology says: “By tone is meant a state of continuous contraction which is slight in extent under normal conditions. It is dependent on the connection of the muscle with the nerve centers. We may assume that under normal circumstances the motor centers are continually discharging subminimal nerve impulses into the muscles, causing changes probably similar in kind to those set up by voluntary effort, but less in degree, the result being that the muscles maintain a state of contraction which, while slight in extent, is continuous. The whole neuromuscular apparatus is in a state of tonic activity and this in turn is traceable back to the sensory impulses continually flowing into the central nervous system and thence switched out by the reflex mechanism to the motor apparatus. It is a distinguishing and important characteristic of smooth muscle that it possesses in a marked degree this power to maintain tone—to remain for long periods in a state of greater or less contraction. The importance of this property is apparent in the physiology of the organs of circulation and digestion. Although it is very unequally distributed among the different kinds of plain muscle, it is certainly important in the physiology of the ciliary muscle.

By contracture is meant a state of maintained contraction or, looked at from the other and, as it seems to us, better point of view, a state of retarded relaxation.

Tigerstedt, page 441 of American edition, describes it thus: "As fatigue progresses and the longer stimulation is kept up, there gradually develops a new condition of the muscle: at the end of the contraction it does not return to its resting position but remains more or less shortened. This condition is called contracture." This contraction is far greater in degree than the continuous slight contraction called tone and is less than the contraction called tetanus. Examples of contracture are common in laboratory experiments on muscles (see Fig. 2). It is not limited, however, to striped muscles. On the contrary, it is more marked in smooth muscles. Less frequently repeated stimuli will produce tetanus in a smooth muscle than in a striped muscle. Similarly, contracture is more easily produced



FATIGUE AND CONTRACTURE (HOWELL)

Fig. 2.—Frog's muscle stimulated fifty times a minute for several minutes. The space between the curves represents the extent of the excursion of the muscle. Initial rise of the upper curve = Treppe; subsequent fall = fatigue. Rise of lower curve = contracture. If the muscle relaxed completely after each stimulus ceased, this curve would be a straight horizontal line. Failure to relax = contracture.

in smooth muscle. The muscle in contracture is not in a state of spastic rigidity, for stimuli applied cause quick further contraction, followed, on stopping the stimulus, by quick relaxation, but this relaxation is only partial, these contractions and relaxations being superposed on the contracture.

What clinical manifestations, if any, are there of this tonus and contracture of the ciliary muscle? They cause latent hypermetropia. The reason the total hypermetropia is not manifest in so many cases is that the ciliary muscle cannot relax and permit the true static refraction to appear. This maintained contraction renders a portion of the refractive error latent. Can we discriminate between the portion due to tone and that due

to contracture? Not always, but it may not be amiss to point out that this is a key to the controversy over how much of the total hypermetropia to correct in our prescriptions. Contracture is an excessive activity and should be stopped by correcting a sufficient portion of the refractive error to relieve the excessive action. Tonus is normal and physiological and does not need to be stopped—indeed, the attempt to stop it is likely to give the patient much annoyance, since the eyes do not take kindly to such treatment and only rarely is it to be advised. Cycloplegics will usually stop contracture and even some of the tone but they cannot be counted on surely to do either of these things. Often an obstinate spasm will not yield to atropin. This is proved when we correct all that is made manifest by the use of atropin and not long afterward find that as a result of wearing this correction a considerable additional quota of hypermetropia has become manifest.

We can now offer an answer to our question, why does the far point come temporarily nearer as a result of continuous strong accommodation? It is a form of contracture or spasm. The ciliary muscle, having been put in a state of strong contraction, does not at once relax; the retarded relaxation is what is defined as contracture. While this explains, in part at least, the behavior of the far point, it is not the whole explanation and it throws no light on the approach of the near point. Before suggesting any other explanation let us review some of the experiments that show the phenomenon in question; we shall then be better able to discuss its nature.

Most of the experiments were made either on ourselves or other graduate students of ophthalmology, ranging from 28 to 60 years of age, while a few were made on children. It is usually stated that more exact and consistent results can be obtained in determining the point of beginning blur by Scheiner's test. In this, as you remember, the test object is looked at through two holes in an opaque disk close to the eye and when the object is out of focus it is seen double. We tried this on a number of subjects but found the results more free from easily occurring errors, more consistent and exact with the natural pupil than with this double pupil. Thus we agreed with Duane, who preferred not to use Scheiner's test as the result of his large experience.

Some tests were made without explaining to the subject what was the purpose of the experiment and the subject was allowed

to keep an erroneous opinion if he had one, till after the experiment was over. It was then repeated after the purpose and expected results had been explained. Thus, in some of our experiments, the subject, in making his observations, was influenced by preconceived ideas in favor of the results, but in others he was influenced against the results, in others as little influenced either way as could be expected. The influence of suggestion could be traced but was found not to be so important a factor as to vitiate results. Pupil measurements were made before and after and sometimes during the tests but after it was determined that the size of the pupil was not an important factor in this particular problem it was not always taken.

The punctum remotum was determined in two ways: first, for distance, by finding the strongest convex or weakest concave lens with which the maximum visual acuity was obtained at 6 meters, both eyes being used together and any regular astigmatism over 0.25 D. or any anisometropia being corrected; second, for near, by using a convex lens over the distance correction and determining the greatest distance at which a suitable test object could be clearly seen. Generally a +2.00 or +2.50 lens was used. With these the punctum remotum of an emmetrope would be 50 cm. when wearing the +2.00 and 40 cm. for the +2.50, if relaxation was complete. Of course it was apt not to be complete, for it is well known how difficult or impossible it is for an observer to relax his accommodation when looking at an object which he knows is near the eyes. Any error due to this cause would be fairly uniform throughout a series of tests on the same subject. The apparent error is larger than the real one, however, for this reason: If a subject accepted +0.50 sphere for 6 meters and +2.50 were added to this and a test made for the punctum remotum it would not be found to be 40 cm., even with complete relaxation, since allowance must be made for the fact that 6 meters is not an infinite distance. An emmetrope could see clearly at 6 meters with a lens of +0.167 D. ($\frac{1}{6}$ diopter), therefore we must add 0.167 to the 2.50 in making our calculation of the punctum remotum. This will give 37 cm. instead of 40.

The head was fixed by a head rest giving support to the forehead and chin, a mouth bit was provided but was found to be unnecessary.

The test objects were of various sorts, such as Duane's hair line (obtained from his authorized maker), Ferree's "li" of vari-

ous sizes, fine print, parallel lines, dots, etc. The test objects were attached to a sliding carrier moving freely on an adjustable optical bench with a millimeter scale. The movement was forward and back, horizontally, along the line of sight of the eye being tested or along the median line in binocular tests. Most of the tests were monocular on the right eye. The test object should not be too small to be clearly seen at the distance being tested.

In an emmetrope the punctum remotum with a $+1.00$ D. should be 100 cm., with a $+1.50$ D. 66 cm., with $+2.00$ D. 50 cm., with $+2.50$ D. 40 cm., with $+3.00$ D. 33 cm. If the tests are made with a small test object which cannot be clearly seen by the subject at a greater distance than 48 cm., the readings would be: with $+1.00$ D., 48 cm.; with $+1.50$ D., 48 cm.; with $+2.00$ D., 48 cm.; with $+2.50$ D., 40 cm.; with $+3.00$ D., 33 cm. This error should be carefully guarded against as it is easily made.

If the punctum proximum was nearer than 12 cm., a concave lens was worn to move it farther out where the variations of a few millimeters would not signify so much when reduced to diopters. Similarly, if the near point was farther than 30 cm., a convex lens was worn to bring it nearer where the test object could be better seen and more accurate answers made.

Another source of error which it took us a long time to eliminate is a peculiar blurring of the image of the test object when it is fixed carefully and continuously even though it is not out of focus. The subject is told to keep the image of the test object clear and shift the position of the test object as may be needed to accomplish this but always keep it as near as possible without blur. It was found that most subjects had periods of blur irregularly recurring and usually very brief. If they tried to follow these and make the image clear by shifting the position of the test object the curve would show great irregularity and the subject would manifest considerable uncertainty. These periods of blur are what Ferree records in his method of measuring ocular efficiency and fatigue. He requires the subject to fix continuously for three minutes a test object, preferably li, and to record by pressing a key to a kymograph the time the object appears blurred and by releasing the key the time the object appears clear; the ratio of time blurred to time clear gives the measure of fatigue. Ferree thinks these periods of blur are due to temporary failure of accommodation. We

feel sure they are not of this nature but are retinal in origin. This does not carry with it a condemnation of Ferree's test, but only of the explanation he offers. If these waves of blur are not of accommodative origin, either they must be eliminated or they will vitiate a test intended to show only blur dependent on accommodation, as ours was. It was found that one of us could fix the test object for three minutes or even ten minutes without any blur to record. This will be taken up more fully in another paper. Suffice it here to say that by directing the subject to keep his fixation roaming from one part to another of the test object these periods of blur could be eliminated. For example, if it were li, let him fix now the top of the l, now the foot of the l, now the foot of the i, then the dot over the i, then the top of the l and so on around, not holding the eye still more than a few seconds at a time. Winking also helps. See charts of tests before and after the subject was shown how to fix (Fig. 3).

Some of the experiments tried were the following:

1. Reading fine print at or near the punctum proximum for various periods up to one hour; punctum proximum and punctum remotum taken before and after; punctum proximum taken at frequent intervals during the test; but punctum remotum not taken during the test, since that would allow the ciliary muscle to relax while it was being tested; muscle balance and "strength" taken before and after. Results: punctum remotum and punctum proximum both were brought nearer, but much less than in the more exacting test (see paragraph 2), where a small test object was steadily focused for similar lengths of time. Focusing does not have to be so exact in reading; we can read tolerably well even if the letters are slightly blurred. Exophoria was diminished and esophoria increased (in one case from 4.5 degrees up to 12 degrees in an hour and right hyperphoria increased from 0.5 degree to 4 degrees in the same time).

2. Focusing small test object and keeping it as near the eye as possible without blur (or it was kept just a little nearer than the true punctum proximum so as to constantly stimulate the eye to make it clear by stronger accommodation), record taken every ten seconds of its distance, tests lasting one, five, ten and twelve minutes and longer up to an hour. Results: punctum proximum and punctum remotum both were brought much nearer (see charts). The effect was almost always apparent in

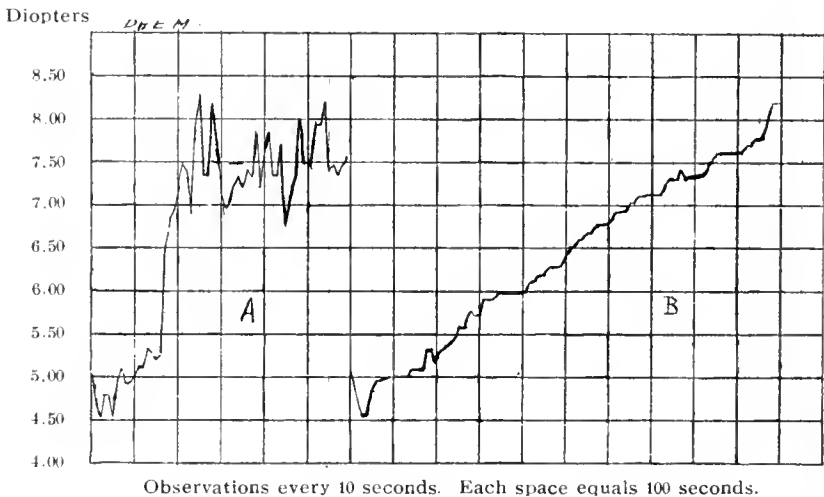
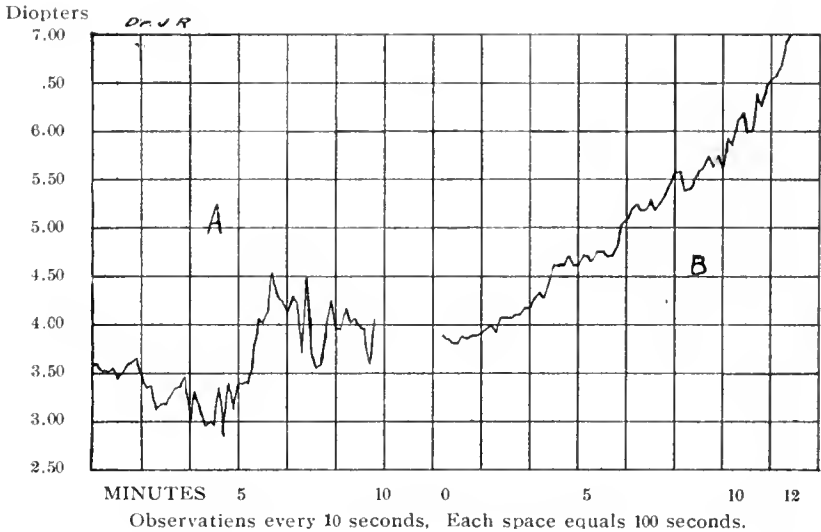


Fig. 3.—Two subjects (both good observers) fixing Duane test object which they are instructed to keep as near the eye as possible without blur. If they get it too near, move it off; if they can do so without blur, move it nearer. Accommodate intently and strongly. The two curves marked *A* were first tests, the subjects fixed the test object as steadily as possible and not simply the object as a whole but some particular point of the test object. Notice the frequent and steep changes indicating uncertainty and a ceaseless effort to secure clear images by shifting the test object forward and back.

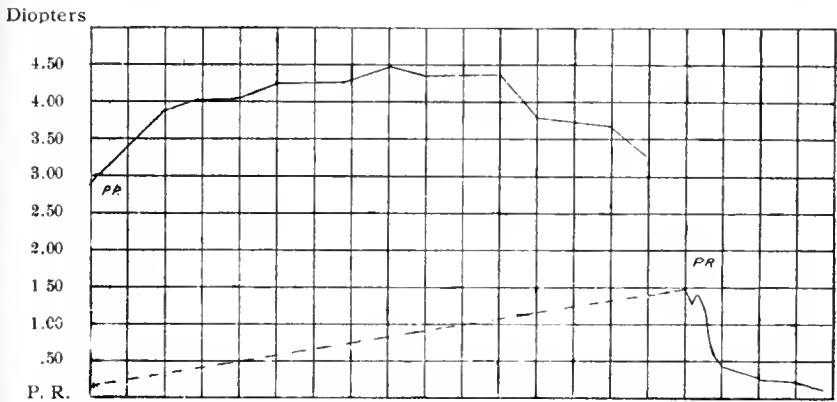
BB, two curves showing punctum proximum in the same two subjects as *AA*, after they had been instructed to keep the eye moving its fixation from one point to another of the test object and to wink freely. They stated, and the curves show, that this caused most of the periods of blur, which were frequent though fleeting in the first test, to disappear in the second so that the curve becomes very steady.

Notice how markedly the punctum proximum is brought nearer, showing a slow but marked increase in convexity of the lens—viscosity. The ciliary muscles probably are not contracting any more strongly at the end than at the beginning. Pupils in the subject of the upper curve were $3\frac{1}{2}$ to 4 mm.; of lower, $4\frac{1}{2}$ mm.

less than a minute, the maximum being reached in less than half an hour.

3. Fixing a test object placed nearer the eye than the punctum proximum time taken until it became clear.

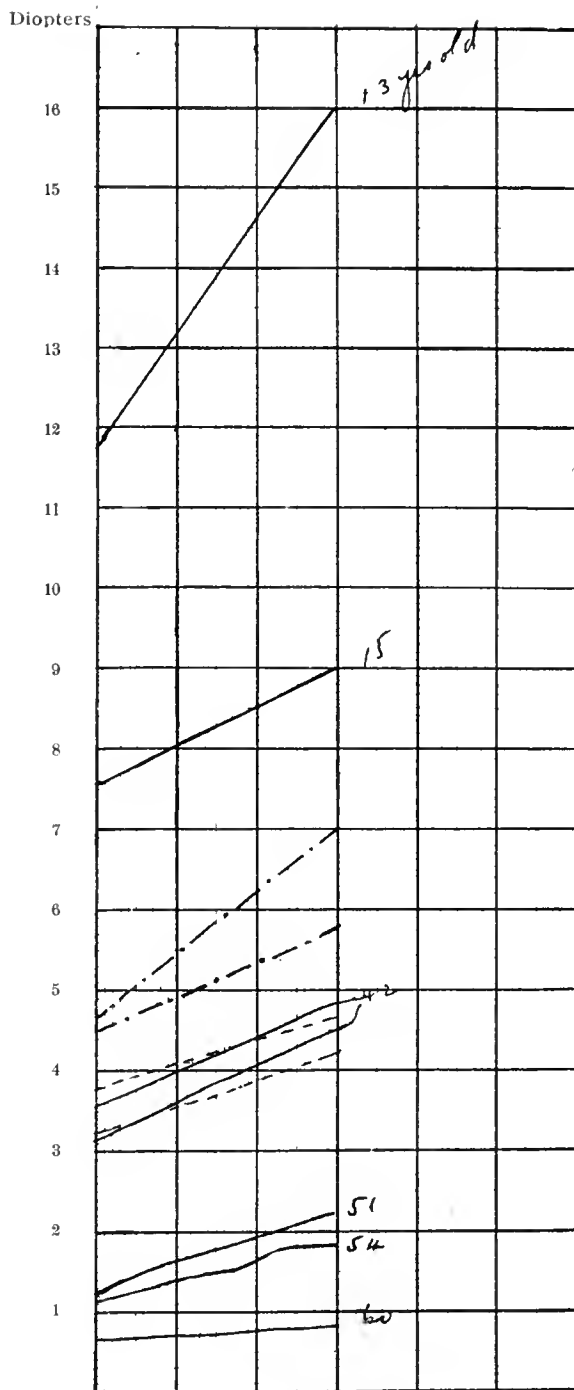
4. Fixing and focusing test object, which was moved rhythmically forward and backward over the range of accommodation from punctum remotum to punctum proximum and back ten or twelve times a minute; punctum proximum and punctum remotum and muscle balance taken before and after the tests. Results: punctum proximum practically unchanged, punctum remotum brought nearer. These results are an excellent corroboration of our explanations of the action both of the punctum



Observations made every 10", but this chart platted from observations every 3 minutes, intermediate observations omitted. Each vertical space equals 0.50 D, each horizontal space equals 3 minutes.

Fig. 4.—Curve of punctum proximum, continuous fixation of test object as near as possible for forty-five minutes, followed immediately by the curve of punctum remotum, continuous fixation of the test object as far off as possible, wearing a +2.00 D. lens. Note the characteristic increase in power to accommodate, followed in this case by a definite falling off after about half an hour—fatigue. The broken line shows how the punctum remotum comes nearer during the very strong accommodation—contracture or spasm. It is followed by the curve showing recovery during the following fifteen minutes, the punctum remotum gradually returning to normal.

proximum and punctum remotum. The ciliary muscle is made to contract as strongly as possible every few seconds, the rhythm was made as rapid as experience showed could be satisfactorily carried out without hurrying. This is similar to the experiment on the frog's muscles and the finger muscles where a stimulus, electric or voluntary, is sent into the muscles at short intervals. It is in such experiments as these that contracture occurs provided the muscle is one of a kind that is susceptible to contracture (not all muscles are). Thus we should expect that the ciliary muscle, if it is susceptible to contracture, would show



5 minutes' continuous focusing at pp.

Fig. 5.—A group of curves of punctum proximum, test object kept as near as possible without blur. The next lowest curve is of a presbyope 54 years old with only 1.225 D. of accommodation at the beginning, but 1.846 D. at the end—a gain of over 50 per cent. The curve at the top is of a lad of 13 years with 11.75 D. accommodation at the beginning, but 16 D. at the end—gain 4.25 D., about 35 per cent. The lowest curve is for a man 60 years old.

it here. Numerous tests proved this to be the case, although most of them were made before we arrived at the theory that this behavior of the ciliary muscle is of that nature.

The action of the punctum proximum must now be further elucidated. Whenever the accommodation is strongly exerted, say for half a minute or more, we have found that the punctum proximum moves nearer and continues to do so for some min-

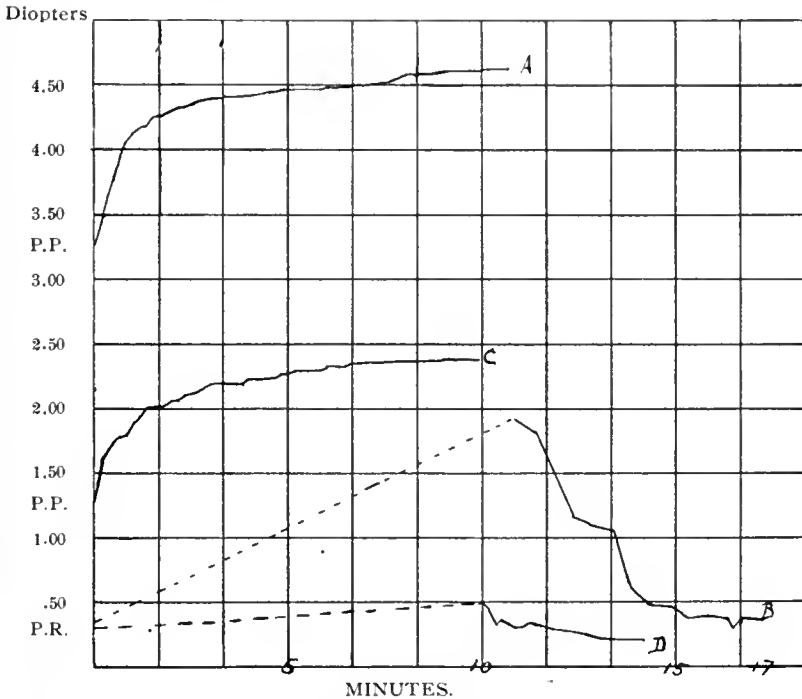


Fig. 6.—This shows the effect of strong accommodation on both punctum proximum and punctum remotum in two subjects. *A*, curve of punctum proximum with continuous strong accommodation for ten minutes. *B* is curve of punctum remotum. At the beginning punctum remotum was about 0.35, at the end of ten minutes of strong accommodation it was 1.90—contracture (inability to relax). From this point the gradual descent of the curve marks the recovery of the punctum remotum as relaxation takes place. In about six minutes after stopping the active accommodation and resting, the punctum remotum has returned to starting point. *C*, same as *A* in another subject nearly 10 years older. *D*, punctum remotum of this subject. It shows less contracture than *B*, viz., punctum remotum from 0.30 to 0.50 and gradual relaxation to 0.19.

utes, perhaps half an hour. We cannot explain this behavior of the punctum proximum as due to an increasingly strong or accumulated contraction of the ciliary muscle, for two reasons: first, because the ciliary muscle does not act directly on the lens but on the suspensory ligament of the lens; second, when the ciliary muscle contracts extra forcibly, we have learned from Hess that the ligament simply becomes more relaxed so that

the lens becomes wobbly. Thus a strong contraction of the ciliary muscle, gradually increasing in force, would not produce the phenomenon we are studying. It would simply make the lens more movable. It is evident that what happens in our experiments is this: The suspensory ligament of the lens being freely relaxed, the lens quickly takes on a more convex shape; the contraction of the ciliary muscle and consequent relaxation of the suspensory ligament being kept up, the lens continues, but at a much slower and diminishing rate, to grow gradually more and more convex. Perhaps its surface does not become more convex; it may be that it is some of its deeper layers that gradually become more convex. It is not necessary at the present time to say just which. We intend to study this question further with an ophthalmophakometer. Now this property of a body of slowly changing its shape under the influence of a stress applied to it is called viscosity.*

The only reference to this property of the lens which we have found is by Schmidt-Rimpler (*Arch. f. Ophthalm.*, xxvi, 103). He was attempting to measure the time required for the eye to change its focus, a matter that had been the subject of investigation by Volkmann¹ and Aeby² before him, and by Barrett³ and Seashore⁴ since. He suggested this factor of viscosity as one element in the problem but not a very important one. It is not mentioned by the other investigators. The factors which they consider and try to measure are the time required for the nervous mechanism and that for the muscular mechanism to act. They found about one second was needed for the eye to change its focus. Undoubtedly the lens has a certain viscosity, as a result of which the changes it undergoes in its shape when acted on by forces applied to it from without or within do not occur instantly or even in a very short period of time; but rather as the force continues to act the lens continues slowly to change its shape. There is an initial very rapid change tapering off rapidly in a slower and slower change, so that at the end of ten

* Strictly and technically speaking the term viscosity is applied only to liquids (*Encyclopedia Britannica*), but in ordinary usage it is frequently applied to solids (*Century Dictionary*) and its meaning in this connection is easily understood. It seems a better term to use than plasticity. Plasticity is the term applied to a body denoting the gradual change of shape which takes place under the influence of a stress which exceeds the limits of elasticity. It is a question for some one well versed in that branch of mechanics to decide what to use.

1. Versuche über die Zeitverhältnisse des Accommodationsvorganges im Auge, *Arch. f. physiol. Heilkunde*, n. F., 1857, i, 17.

2. Accommodationsgeschwindigkeit d. menschl. Auges, *Ztg. f. rat. Med.*, iii Reihe, 1861, xi, 300.

3. The Velocity of Accommodation, *Jour. Physiol.*, 1885, vi, 46.

4. On Monocular Accommodation-Time, *Studies from Yale Laboratory of Psychology*, 1893.

minutes the change has become so slow as to be only just noticeable when tested at intervals of a minute or more.

The experiment discussed above, in which the test object was moved forward and back over the range of the accommodation ten or more times a minute ought not to show a tendency for the punctum proximum to come nearer the eye, if this theory is correct, because a continuously acting force is necessary to produce a "set" in a viscous body and in this experiment the action of the force is as far as possible from continuous in one direction, being reversed every five seconds or so. It is all the more convincing that the experiment was designed to show something else.

The nearest approach to discovering this phenomenon seems to have been the observations on eyes under eserine. Here the ciliary muscle is strongly contracted and the zonule completely relaxed, consequently the punctum proximum should be brought nearer than it was when the examination was made in the usual way before eserine was instilled. This is just what was found by numerous observers. Various explanations were offered. After Hess showed that less than this maximum contraction which we get under eserine would suffice to relax the zonule, the old and natural explanation that the increase in accommodation was simply due to stronger contraction of the ciliary muscle had to be dropped. Voluntary contraction far short of this maximum is sufficient to fully relax the zonule. How then is the marked approach of the punctum proximum to be explained? Hess tries to explain it as simply an *apparent* approach. The actual focal length, he thinks, is not less than before eserine was used, but the very small pupil makes it possible to see with the small circles of diffusion clearly enough to deceive the observer into thinking his eye is focusing the test object when really the object is well inside the actual focal point. This explanation will not account for the approach of the punctum proximum which we observed, because we did not use a miotic and the change in the size of the pupils was too small to be a factor of importance and the course of the observed changes in accommodation did not by any means follow changes in the size of the pupil at the same rate.

The question may be asked, how large are the quantities being measured in these experiments and what are the limits of error? Perhaps if the margin of error is considered it will be found that the observed changes in the range of accommo-

dation are not larger than can be accounted for as mere errors in observation. We found, of course, that if we took a series of readings of the punctum proximum or punctum remotum (and we always did take a good many readings with every subject examined) they would vary more or less. The subject often said that he could not set a definite and sharply defined spot as the exact focus. If you try determining the focal length of a lens by measuring how far a screen must be held from it to produce a clear image of some distant test object, you will find, especially with a small pupil or stop, that there is difficulty in locating the exact spot and if half a dozen readings are taken there will often be considerable variation.

Practice reduces this a good deal, but it cannot be entirely eliminated because it is inherent in the nature of the phenomenon being observed. After reducing the readings in our experiments to diopters we found a variation of over 0.25 D. was rare. Often a series of four to six readings, taken in quick succession, would not vary more than 0.01 D. Large variations were classed as errors and treated accordingly. If the measurements had been made by the subject holding the test object in his hand and measuring the distances in the rough way in which the punctum proximum is determined with sufficient accuracy for ordinary clinical purposes, probably the margin of error would have been so wide as to vitiate many of the readings. An analysis of our observations with this in view showed that the changes in the range of accommodation both in punctum proximum and punctum remotum were greater than the margin of error in the observations. Thus the errors in observations would average less than 0.1 D., while the changes in the range of accommodation were several times as large and therefore could not possibly be accounted for as errors of observation. Moreover, they were consistently in the same direction, while errors of observation vary both ways around a mean. In some cases where the changes in the range of accommodation proved disappointingly small we found that measured as a percentage of the total range they were surprisingly large and strongly corroborated our theory.

APPLICATIONS

Any thing that throws light on the function of accommodation is sure to be of practical use to us, because when one has mastered the peculiarities of accommodation under all conditions of refraction, age and state of health, he has gone a long

way toward a complete mastery of the most important chapter in ophthalmology—the measurement of errors of refraction and their treatment.

The results of the experiments which we are reporting seem to emphasize two important and opposite sides of the problem:

1. It takes very little active stimulation of the muscles of accommodation to produce a contracture. As a result, the eye will not accept the full correction of its error of refraction until the contracture has subsided. Therefore it is of advantage to instill a cycloplegic so as to eliminate this source of error. This is in addition to other advantages which accrue from using a cycloplegic. It prevents those efforts to accommodate which are so easily set up by the mere looking through a trial frame at a test object and trying to make out letters or lines at or near the limit of visibility. The result of this effort is that the eyes show preference for lenses which are weaker plus or stronger minus than are best for the patient, simply because when the patient strains to see, he exerts his accommodation and so sees better with a lens which permits or encourages this accommodation. Using a cycloplegic checks this tendency. Unless these sources of error can be avoided in some other way, a cycloplegic is essential to reliable results. We believe that they *can* be avoided in the great majority of cases by employing methods which it would take too much time to elaborate here. What we wish to point out is that unless one finds, after an adequate number of test cases, that his results when testing patients first without a cycloplegic and then with, agree closely and consistently, he should use a cycloplegic. This, by dilating the pupil introduces other sources of error so that absolute agreement of results with and without a cycloplegic is not to be expected and would be suspicious if reported. This is why results obtained with a cycloplegic must be gone over and compared with results without a cycloplegic before deciding on proper treatment.

2. On the other hand, our results show that even after prolonged contraction of the ciliary muscle to the maximum voluntary degree, the resulting contracture rather rapidly subsides if the patient stops accommodating, especially with the eyes shut. We believe that in this we have a very important and useful trait and that advantage should be taken of it in our practical work much more than is customary. We have found it exceedingly valuable and have made it a studied feature of our clinical methods for years. In this we were doubtless influenced,

one of us, by his first teacher, Mauthner; the other by his teacher Snellen, Sr. Mauthner was the first to point out that latent hypermetropia disappears when the patient directs his eyes at the distance in the dark room without any fixation object, and so the total hypermetropia can be determined by the ophthalmoscope by the direct method. Mauthner was not only a good observer, but he was myopic — 6.50 D., and so was at an advantage in estimating refraction by the ophthalmoscope. He says (*Optische Fehler des Auges*, p. 285) that though on the watch for years for an exception to this rule, he had found but one case (and that a strabismic) in which the refraction as measured by the ophthalmoscope showed less hypermetropia than by subjective tests with lenses. His teaching was not without effect and many of the greatest authorities, especially European, have followed him in this. Hess repeatedly quotes him on this point (relaxation in the dark room) with approval in his monograph on refraction, which is the last word from Germany. We do not wish to imply that we are satisfied with the degree of accuracy in measuring refraction which is attainable by such rough approximations. But when due allowance is made for its limitations we must admit that the conclusions of such observers are well worthy of consideration. We have no doubt that there is an important but neglected truth in the teaching of Mauthner, that the ciliary muscles relax their excessive contraction when they are in the dark room and are directed to the distance without fixation object.

We do not believe that the whole of the latent hypermetropia will become manifest in all cases in the few minutes that the patient is in the dark room for ophthalmoscopic examination; but then neither will the whole of the latent hypermetropia become manifest as a result of instilling atropin in all cases. We are sure that something can be gained in securing more relaxation of the tonic contraction of the ciliary muscles and of the "contraction remainder" (Starling Physiol.) or contracture by removing all possible stimuli to accommodation for some minutes before examination and as far as possible during examination. Maddox, than whom there is no more trustworthy teacher in England, gives this advice (*Golden Rules of Refraction*, p. 46): "Bid the patient close his eyes while the convex lenses are being changed, to avert an act of accommodation." We had independently discovered the value of this procedure and have followed it and taught it for years. Again

Maddox says (p. 47): "A very good expedient for discovering latent hypermetropia is to make the patient shut both eyes before placing a pair of weak convex lenses before him. On opening the eyes, I find they will often accept a fuller correction than otherwise." Another good expedient (Hartridge) is to commence by an overcorrection of the hypermetropia and work down. Both these expedients recommended by Maddox are well known. They are quoted simply because they are applications of the principle to avoid anything that allows, much less requires, the patient to accommodate even for a moment. For this reason we avoid taking the patients' visual acuity without glasses as the first step in the examination, particularly if they are hypermetropic, since it encourages them to accommodate. This is advised also by Paterson and is approved by Maddox. Beginning with the examination in the dark room, which is the darkened examining room not a small closet, lenses which somewhat overcorrect the hypermetropia as roughly determined by the ophthalmoscope are placed before the patient's eyes while still in the dark so that when he opens his eyes after the light is admitted to the room he will have no inducement to accommodate. During this time in the dark, especially if he has not had to move from his seat and has been mentally at ease, his ciliary muscles have relaxed and by skilful management can be kept relaxed long enough to determine astigmatism and hypermetropia in most cases without a cycloplegic. As stated above we do not advise any one to dispense with cycloplegics until he has found by experience that his results warrant it. Then he may reserve cycloplegics for exceptional and selected cases. It is not very rare for the application of these methods to bring to light latent hypermetropia which homatropin or even atropin had failed to reveal. If there is much contracture it will be necessary for the patient to wear the glasses some time. This also aids the nerves to relax when there is spasm, and this is more important than the contracture, but both should be reckoned with.

The following common experience illustrates both these factors and the importance of avoiding accommodation in our methods of testing refraction. If a patient is examined with due care to avoid accommodation according to the plan suggested above or some similar method and the strongest convex lenses found with which maximum vision can be obtained (suppose it to be 6/4 in a hypothetical case with 2.00 D. sphere and less

than 0.25 D. astigmatism, O. U) and if the glasses are then removed and the patient allowed to use his eyes a few minutes, after which the same lenses are put on and vision taken again, it will often be found that vision is materially lower now (say 6/6) and is only brought up to its previous standard by using a weaker + sphere (say + 1.75 or even + 1.62) or by shutting the eyes for a few minutes or by wearing the stronger glasses some time. This is because the nerves and muscles having been allowed for a few minutes to take on their habitual amount of activity, they do not readily relax, even in the interest of good vision. In part this is the fault of the muscles (contracture), in part it is the fault of the innervation. As Hess says: "Relaxation does not take place at once in eyes with hypermetropia when convex lenses are placed before them, because the requisite innervation is quite unaccustomed (*ganz ungewohnt*), but if the glasses are worn continuously the relaxation follows more or less rapidly."

The following is an instance of the effect of viscosity as we often see it in practice. We have all had patients tell us that after reading or other near work they find on looking up that it takes an appreciably longer time than it used to, or as they think, than it ought to for the eyes to adjust themselves to distant objects. Usually these patients are insufficiently corrected hypermetropes or presbyopes, that is, at near work they have to exert their accommodation more than normal. Two things follow: (1) A certain amount of contracture which causes a temporary myopia, so that when they look up it is not until enough relaxation has taken place that they can see clearly; but also (2) the lens, after prolonged accommodation, does not at once reach its proper form when the accommodation is relaxed, because its viscosity prevents rapid complete change of form.

DISCUSSION ON PAPERS OF DRs. NEWCOMB, WOODS AND LANCASTER, AND WILLIAMS

DR. ALLEN GREENWOOD, Boston: We are much indebted to Dr. Woods for taking us into his confidence and showing us so intimately the man and his work. Such papers as this are invaluable. He says eyes are the receiving end of a toxic irritation, and this at once opens up the whole subject of the effect of bodily conditions and functions on the integrity and usefulness of the eyes, and how this emphasizes the fact that ophthalmology cannot be divorced from general medicine and points to the fact that all should have some practice in general medicine before finishing their ophthalmologic training.

The main object is the comfort of the patient and the placing of his eyes in the best condition for the daily work required. This does not

always mean we must determine the absolute static refraction in every case. I agree this is necessary in the majority of cases and should be obtained in all young children. In adults under presbyopic age I try the ophthalmoscope, retinoscope, trial case by the fogging method or Verhoeff's method, and if they yield consistent results I order glasses. I often have patients close their eyes for fifteen minutes and then I reexamine them. If I can obtain absolute comfort with glasses for my adult patients without the use of a cycloplegic I do so, though this does not always mean that the patient may not have to come back later on for a cycloplegic examination. We have many adults in whom the use of a cycloplegic is a disturbing element in their lives and if you can without it carefully follow out the tests and attempt to prevent the undue accommodation and spasm by the above-mentioned methods and get the desired results, it is sufficient.

Then, in regard to the muscle tests and the treatment of the muscles that Dr. Woods has outlined, I do not believe exercise will do much except for the internal recti, as I have tried to influence the elevators and depressors of the eye with little influence. The careful working out of the refractive anomaly is the secret of obtaining muscle balance in almost all cases. I seldom order prisms unless base up or base down.

The use of homatropin to rest the patient's eyes at the end of the week is valuable. I have not tried that but have used mydriatics for a few days at a time, and in some cases atropin for a week at a time in case of ocular spasm, particularly in myopes, and it is sometimes the only way in which we can make them absolutely comfortable.

Dr. Lancaster has shown us in a clear and scientific manner what the result of a cycloplegic is and why we use it and why we do not in some cases where we might ordinarily expect to use it. The more such scientific work as this can be done in this country the better.

DR. EDWARD JACKSON, Denver: The paper of Dr. Lancaster and Dr. Williams is certainly very interesting, and one that it is very satisfactory to have presented to the Academy. They open up lines of investigation that we can all follow for ourselves, to test the results which they report and to form our own conclusions as to their explanations of those results.

I have observed, as a rule, that the patient will give at first a near point that is too far away from the eye. But persisting for half a minute or so in trying to get the patient to see nearer, he does so; and perfectly distinctly to a point decidedly nearer to the eye. But attempting the same thing with my own eyes I failed to get a near point other than that arrived at in the first very few seconds. The explanation in my own mind, which may be incorrect, has been that many patients making the first attempt do not put forth the effort that is necessary to bring out their full accommodation. But if you keep them trying they presently do more than they did at first. Whether it is a muscle phenomenon, or whether it has to do with the nerve centers being able to discharge a higher amount of nerve force later than at first, is uncertain.

With reference to the far point: We have all observed that at many times people are slow in regaining their true far point after near work. I think an element that is not referred to in the paper, but which is important, is the element of age. It is unusual to observe this delay in young people, but rather common to observe the failure to relax their accommodation in people who have reached the age of presbyopia. This might be due to a change with age in the physical properties of the lens.

The term "viscosity" is a good suggestive term, but I doubt whether the property indicated by it in the paper differs from elasticity in gen-

eral. For a body to assume a position or shape by means of elasticity always requires a certain amount of time. If there is an unusually long time consumed in the change in the form of the lens, it has a "slow elasticity."

We must not forget that we are also dealing with an unstriated muscle, which does not seem to be an apparatus adapted to very rapid change of dimensions. It cannot change shape so quickly as striated muscles. Taking the length of the circuit into account, we find the reaction of the pupil is slower than the reflex actions of our voluntary muscles.

DR. FRANCIS VALK, New York: I can well appreciate the vast amount of work Dr. Newcomb has accomplished in the examination of these one thousand cases of refraction under full cycloplegic and I have been much interested because of the privilege of comparing his results, according to the abstract of his paper, with some of my own made nearly twenty-five years ago. In those days we had very few scientific means of examining the refraction by the objective methods and were compelled to use a cycloplegic to determine what was the exact refractive condition of the eyes. The ophthalmoscope was the best objective means at our command. We began to use the retinoscope about this time and I well remember the day when the late Dr. George Bull of Paris showed me a little piece of looking-glass with a hole in the center to look at the reflex from the retina, as the light was thrown into the eye at a distance of three feet. Also in those days St. John Roosa advised the use of that excellent instrument the ophthalmometer of Javal, by which we could measure the radius of the corneal curve and so estimate, very nearly, not only the amount of the astigmatism and the axis of the same but also estimate the probable refractive condition of the dioptric apparatus, as I stated in a paper in 1897, on the relation of the curvature of the cornea to the refraction of the eye. These exceedingly valuable methods have come to the oculist of to-day and his methods of examination of the refraction are almost complete; so much so that I stated in this city, at a meeting of the A. M. A. in 1906, that I expected the day would come when we could prescribe our glasses without the subjective examination, in other words, entirely independent of the statements of our patients. But that day has not yet arrived and we must question our patients as to the acuity of the vision; yet, in my own work, I pay no attention to their answers unless they fully agree with my objective examination. If then I return to my work of 1892 I find a vast difference in the percentage in these refractive cases, which shows the great advantage of our present methods. In my records made at that time hypermetropia stands at the head of the list with 35 per cent., almost the same as Dr. Newcomb's record of 34.1 per cent. of compound hyperopic astigmatism. This shows an undoubted advancement, as it is evident that in my early work, depending on the subjective examination, I failed to find many cases of astigmatism and only noted the hypermetropia. At that time I did find 16 per cent. of compound hyperopic astigmatism and 52.2 per cent. of my one thousand cases shows astigmatism of some kind. I cannot understand how Dr. Newcomb finds such a large number of cases of mixed astigmatism. My examinations to-day show the same percentage as in 1892 and I do not consider that the refractive condition of mixed astigmatism is subject to much change by the accommodation; in fact, in my estimation, it is due wholly to the curve of the cornea and that the anteroposterior diameter of the eyeball is the same as in emmetropia. I know that these figures are very interesting and this paper shows some advance in the examination of the refraction, but I cannot agree with him when it is said that the "methods of examination are inadequate," as his records show how

complete they have been; yet I must ask the question, why use full cycloplegia? A careful and proper examination in children and young people will always show the correct refraction unless complicated by conditions that demand atropin. I seldom use it, as I find that the retinoscope and the ophthalmometer with the ophthalmoscope will always reveal to me the true refraction, except in very old people, whose pupils are small, and in these cases we do not need a cycloplegic anyway. Make your subjective examination agree with these three tests and you will find the glasses needed without atropin in the vast majority of your cases.

DR. LINN EMERSON, Orange, N. J.: Dr. Newcomb speaks of the unreliability of homatropin, but I find it very reliable. As a result of a series of cases in which I used it on forty patients, in seventy-seven out of eighty eyes the refractive condition was the same. Every instillation was made by my own hand and thirty minutes allowed to elapse after the last instillation, and I have not since given it to them to take home and use themselves. In regard to prism exercise vs. prisms bases in, there are many cases in which the exercise relieves patients temporarily, but as soon as they are better and discontinue the use of the muscles, they go back. In two years or so they return with identically the same condition, and the test generally shows the same amount. I do not see that they are done any harm by giving them two-degree prisms, base in, for constant wear and their exophoria is generally not increased. They return five or six years later with no greater exophoria than in the beginning and they are comfortable. When they have esophoria for distance and exophoria for near I find they really do very comfortably and satisfactorily.

DR. E. E. HOLT, Portland, Me.: I realize what an immense amount of work these papers represent, for I am making statistics of thirty-five thousand eye cases recorded in private practice. I have a form of my record book here showing my method of keeping records, which I will pass around for inspection. When I began the practice of medicine forty years ago I could not find any specially devised record book, so I devised one. I have been making slight changes in the different editions until I have reached the one I present here. At the American Ophthalmological Society thirty years ago there used to be considerable animated discussions about the necessity of using a cycloplegic in testing eyes for lenses, one group contending there was no need of it, while the other was equally positive that it was absolutely necessary in order to determine the proper lenses to be worn. I used about a 1 per cent. solution of atropin for years for the purpose of determining the amount and character of ametropia and the lenses best suited to correct it. I was, of course, obliged in some cases to determine what lenses I would give without the use of a cycloplegic. This practice led me to determine just exactly what lenses I would give in each case if I could not use a cycloplegic in order that I might compare those lenses with what lenses I would give after I had used the cycloplegic. This experience taught me to get along without the use of a cycloplegic in a very large majority of cases. I early began to examine the eyes of my ear patients and found just the same amount of ametropia among them as among the eye patients. I found the correction of this ametropia one of the most potent remedies for the relief of tinnitus aurium.

DR. CHARLES MAY, New York: Dr. Reber refers to the handling of the patient; the manner in which this is done makes a wonderful difference, not only in regard to results but also in the time consumed. The patient is seated in front of the chart and he is not quite sure what is expected of him. He may think that he is expected to study out the test-letters or else that he is to read only those letters of which he is certain. If he is

allowed to dwell on the letters any length of time you will be testing his vision *plus* his judgment, or his vision with results altered by his judgment, and his judgment may mislead both you and him. It is well to say to the patient: "Give me the names of the letters quickly; do not stop to analyze since, if you do this, you are altering a visual impression by mental speculation; I do not want to hurry you since I am in no hurry myself, but unless I get your first impression of each letter quickly, before you have time to change it through using your judgment, you will not give me the best possible information. Let me hear what you see at first glance." You will thus gain an enormous amount of time because you will shut out much extraneous and immaterial and often silly comment on the test-letters and the patient's ability to read them. You all know how fond patients are of telling you a lot of stuff which has nothing to do with your estimation of his acuteness of vision and the subjective examination. But if you prepare the patient in the manner just indicated the subjective results will correspond more closely to the objective findings.

DR. WALTER B. PARKER, Detroit: A case of iridodialysis came under my observation some two years ago on which could be demonstrated the work done by Hess. The periphery of the lens could be plainly seen. By having the patient fix for far and near, the lens could be seen to settle down. It took several seconds for the change to take place. Whether the change in position was due to viscosity of the lens or to slow action of the ciliary muscle, I am unable to say.

With regard to the treatment of exophoria, I may be somewhat old-fashioned, but I still think the operation of tenotomy the best method of treatment in cases of exophoria which persists after the power of adduction has been fully developed.

DR. H. GRADLE, Chicago: Dr. Lancaster asked me to discuss his paper, but the only possible discussion is on the type of measurements used. The results were so carefully observed that another type of measurement might be valuable as a check. Possibly he might use the third image of Purkinje, because the changes he wishes to observe are probably in the curvature of the lens.

DR. F. H. VERHOEFF, Boston: I should like to ask Dr. Lancaster whether or not he has excluded the possibility that the increase in refractive power to which he refers may not largely have been due to the action of the extra-ocular muscles, resulting from the great effort to accommodate. This possibility does not seem to me probable, but I think it should be excluded.

DR. WOODS (closing discussion): I agree with Dr. Emerson's criticism of the muscular cases that come back with relapse, and the necessity of repeating exercises. They do come back and these exercises have to be reverted to indefinitely. It is a vicious habit the patients have formed. Prism exercise corrects it for a time; that is all. As the old tendency reasserts itself, more exercise is needed. The only exceptions are young persons. They sometimes keep well under refraction correction.

Dr. Reber's remark about the girl who developed internal squint and manifest hypermetropia from overcorrected myopia, in spite of atropin, would be true if it were so, but it is not. When discharged with concave glasses she had a very low grade of esophoria.

I endorse all that has been said about the difficulties of subjective tests. The idea of having the patient shut the eyes frequently is excellent. Get him to give his first impression from glasses and not study things out. "Shut the eyes and start over again." I suppose I repeat this a dozen or more times a morning. One must use judicial rudeness sometimes,

especially with cultured women, to get them in a proper state of mental inertness.

DR. W. REBER, Philadelphia: It will be generally admitted that skiascopy is a good and faithful servant. I have employed it twenty-two years myself and should regret to be deprived of it; but when it comes down to the last quarter of a diopter of error I do not hold myself responsible. I use the retinoscope many times every day; but the uttermost refinement as to result depends on how good ophthalmologists we are; for the best ophthalmologist is the one who quickest trains the patient as to the part he is to play in the little drama. Dr. Wood's case probably had exophoria. The thing that is of value is the study of the convergence; of more value than any prism adduction. The latter can be made anything you choose. Of great value is the parallax test which was first devised by Alfred von Graefe and popularized in this country by Duane. Normal muscle balance I should like to talk about for some hours. In my judgment we should coin a new term. It should be known as euphoria. Orthophoria we have come to accept as that condition in which there is absolute or mathematical balance for both lateral and vertical muscles. The people who are most comfortable with their eyes are those who present about two degrees of esophoria for distance and two to three degrees of exophoria for the near point. Anyone with much experience with abnormalities of muscle balance will, I am sure, have come to this same conclusion. For this state of the muscles the term euphoria would be acceptable, and I am hoping it will come into general use.

The laboratory work done by Dr. Lancaster and Dr. Williams shows what can be done with simple apparatus by those who wish to follow it up. The extreme relaxation of the zonule to which they refer has given me much thought. A number of times I have had a patient accommodate to their fullest extent and have then studied the eye under binocular magnification but in no instance have I been able to find the extreme relaxation of the zonule producing what was practically an iridodonesis. However, this is a phase of the subject which needs to be carefully followed up by all of us. Concerning the matter of fixing an object it is well to remember, as Dr. Dodge has shown, there is no such thing as absolute steady fixation. His photographic records of ocular movements prove this beyond all peradventure.

DR. LANCASTER (closing discussion): Regarding technic, precautions, etc., I ask any who are interested to come to the laboratory at the Harvard Medical School to-morrow and see how the experiments were done. I am interested in Dr. Jackson's suggestion that the apparent approach of the near point may be psychological. This is precisely what we thought at first and only by degrees were we willing to admit that we found something else. I should like to have our methods criticised on the spot.

Dr. Gradle and Dr. Verhoeff suggest very properly that objective tests confirming our subjective findings would render them more convincing. In our paper we speak of that as something we propose to do. This report is only preliminary; there is more to be done; we expect to pursue the matter further and hope others will.

STRABISMUS

FRANCIS VALK, M.D., Sc.D.

NEW YORK

"Within the lifetime of a single generation, the treatment of asthenopia, presumably dependent upon faulty ocular conditions, has assumed a complexity not fully comprehended by our immediate predecessors."—Risley, *Ophth. Rec.*, July, 1912.

It is possible that I am carrying coals to Newcastle when I decided to place this paper before the profession, but during many years of observation in this field of ophthalmology two questions have often come to mind for which I can find no solution as I read the literature on this subject. I do not think the true cause of strabismus has been fully explained, nor do I believe the last word has been said, for if I ask what is the true or primal cause of a deviation of the optic axes, that is to say, what abnormal element is found in all cases, I find no answer in our text-books of to-day. And again, I ask why squint disappears, in some cases without glasses and in some with glasses. Finally, I would like to ask the question, even though we are urged not to operate until all other methods fail, when should we operate on these cases, or to put the question in another way, should we not operate on all cases of squint? That last question is a very broad one, and I fear many will say it is all wrong; but it is a question often in my mind, though I have not yet the courage to urge its advancement.

I am not willing to accept any of the theories that have been advanced during the past twenty years, in fact, since the days of Donders' antithesis, which tells us that "hyperopia causes convergent squint, and myopia causes divergent squint," although this was one of the first theories given to me in my early work in ophthalmology. This theory, to my mind, has disappeared long ago, to say that convergent or divergent squint may be due to many other causes, one from one prominent symptom, and the other from another prominent symptom, seems to me to make the question very complicated and does not answer any of my previous questions. Some eyes converge, some diverge, some have one eye up, the other eye down, and finally some have both eyes up or both eyes down (double squint). All these deviations have been explained in many ways, but they are all abnormal positions of the optic axes, and consequently may we

not say they all have the same common cause for the deviation? I believe so and trust this paper may prove my contention.

To return, then, to what I have said, I ask, why do some children squint, and in time the optic axes become parallel and remain so? We have histories of these cases. Another child squints, yet, with glasses correcting the refraction, we have obtained the correct position of the optic axes. About 20 per cent. of all cases seem to come under this classification. Finally, another child must have an operation on the ocular muscles, as all procedures which seem to have corrected the deviation in the other children now fail completely, and the surgeon must decide on an operation after months of trial to correct an abnormality. Again I ask, if this operation could not have been decided on at once without this attempt to correct the squint by other means?

All these questions are interesting, and I have endeavored to find the reason or, in other words, the diagnosis between the child who does not need an operation and the one who finally must have it. Now, obviously all these children presented the same inward or outward deviation, generally the left eye turning in about 80 per cent. in convergent squint, and the right eye turning outward with the same per cent. in divergent squint. Again, nearly all the cases of convergent strabismus are found to be hypermetropic, and we are enthusiastic over each case which seems to have the eyes paralleled with glasses, and say hypermetropia is the cause; yet our next case completely fails with the correction of the hypermetropia. Furthermore we may find a loss of fusion force, the antipathy to single vision, and now we have another cause for squint, and so we look for amblyopia, fusion force, accommodation, myopia, etc., to account for the deviation. But at last, when we decide to operate, we have only one condition, namely, the ocular muscles.

To-day is the age of progression in all branches of medicine, and our men of initiative are now working along these lines in every condition of the system apart from the normal in which they find, or endeavor to find, some specific cause. That is to say, all disease must be due to some specific micro-organism, which if found and eliminated must remove or cure the disease. Can we not say then, that any and all abnormal deviations of the optic axes must be due to some special cause that may be demonstrated in every case? This may be, and often is, complicated by other predisposing conditions that are more pro-

nounced than the primal cause. Granting this, it seems to me one can make the statement that in all cases of squint we have a deep or primal cause, but so complicated that the correction of a predisposing condition may correct a deformity, though the true cause remains the same.

The above proposition is what I purpose to explain in this paper. To find an answer, we must first have a question; and so we ask, what is the ever-present cause of strabismus? and again, why are some cases so quickly corrected by glasses and others, similar in all obvious conditions, must demand an operation?

I have studied, considered, and applied the many and various theories which have been advanced since the days of Donders, and I think my work has shown the usual conditions of success. But as the work has proceeded I have seen many cases that seem to prove beyond any doubt that all these theories, even the wonderful one of fusion, must have some underlying cause, and that other writers have based their conclusions on some few successful cases and dismiss the others by a discreet silence. To say that hypermetropia causes squint, and that the next case we see of convergent squint is myopic, seems to me against Donders' theory. Again, one case of squint has perfect binocular vision as soon as we correct the refractive condition; and I have seen a case of congenital amblyopia without squint which had excellent fusion power.

These cases seem to prove the fallacy of the theories of fusion and of amblyopia. Furthermore, we meet cases of divergent squint with a very evident refraction of hypermetropia. Even though myopia seems to follow divergence, yet it does not predominate in exophoria; hence myopia cannot be the true cause of divergence. These are the conditions that to my mind bring up the perplexing question as to why children or grown people squint. This has led me to look for some condition, anatomical, that was, and could be proved to be, present in each and every case. I have stated in my earlier writings on this subject that there must be one underlying cause, which may not be sufficiently developed to produce the deviation, unless complicated by these many other causes, now called theories; or those cases in which the primal cause is fully developed and in which the correction of all other theories will result in failure until the primal cause is corrected, that is to say, until some operation has been performed. If this is

axiomatic, then we may say as a corollary to that theory, that if an operation must be performed, it being the final procedure, then the reason for that operation must be the true cause of all cases of strabismus. We are told that the asymmetry of the orbits may cause squint, but we do not change the contour of the orbits when we operate on the ocular muscles correcting the deviation for the rest of the individual's life. Yet, if the asymmetry caused the squint, why does it not return? Furthermore, it has been asserted that all latent squint was due to muscular conditions, but where shall we draw the line between latent squint and fixed squint, so far as these before-mentioned complications are concerned? Only the latter condition is obvious; still all the anatomical conditions are the same in both.

It seems to me that all these questions may be answered without argument. We must take into consideration all these theories, and not accept any one theory, unless we find that some one condition exists in all cases of strabismus, and then its correction should produce the desired effect. May I not say that all these other theories are merely contributing causes to one that is ever present? This argument seems to imply that all cases of latent and fixed squint should have an operation. While I have not fully arrived at this conclusion, I am much inclined to think so, for reasons which will be advanced in this paper. If we establish a theory we must prove its existence, and then, applying our correction to that abnormal condition, will our procedure be a success or a failure? I venture the assertion that all our operations for squint should be successful, but I find these negative questions advanced by Bielschowsky, in an interesting review by R. B. Hird, in the *Ophthalmoscope*, July, 1912, on "Operative Failure of Squint."

He classifies the cases of squint in three groups: First, the fully successful cases, where binocular vision is obtained. This is possible when there is no amblyopia or congenital defect of fusion. Second, a large number of cases only partly successful, cosmetic result good, without binocular vision (generally with amblyopia); and finally, group three, the unsuccessful cases. While the writer may agree with the propositions set forth in these three groups, yet in his personal experience he must doubt the fact of the third group, the unsuccessful cases.

It goes without saying that we do see many cases of strabismus whose history tells us that they have had one or more operations on the ocular muscles, and yet the deviation of the

visual lines, or optic axes, still remains; and as Bielschowsky well says, "Faulty operative technic may be the factor." But not "may be"—is it not always so? And may we not state that all cases of strabismus should and can be made successful? I am inclined to take this view, as from my past experiences, I object to the statement, "operative failure of squint"; and if I may be allowed to so state, I would prefer to say operation not a failure, but the operator's judgment was at fault, thus producing the failure. To go back to the old antithesis of Donders, and to suppose that hypermetropia causes squint, and to operate with that diction in view, would be very apt to cause "operative failure"; for if the refractive condition causes the strabismus, then why operate on the muscular apparatus? To do this, as I have already stated, seems to me to violate one of the cardinal rules of surgery, for to be successful one must operate on the cause, the *fons et origo*, of the deficiency.

Furthermore, is it rational to assert that hypermetropia causes convergent squint and myopia divergent squint, when we cannot correct these deviations of the optic axes without an operation on the ocular muscles? Unfortunately for the proving of our argument, and perhaps because the eye is not a mechanical or material optical instrument, but rather a part of the brain, we do find certain children in which the deviation seems to disappear under the use of hypermetropic glasses. I state "seems to disappear," as I am fully assured that the primal cause still remains, as even this temporary correction will not apply to convergent squint in which we have a refraction that is myopic. Hence we must concede that there must be some other condition existing in all cases of strabismus, and that in many cases the hypermetropia is a strongly contributing cause, great enough to produce an overbalance in a case whose true or primal cause is not very pronounced. I shall refer to this later. But to return; if we have failures, let us first inquire what should be considered successful cases. Here I may make the broad statement that all operative cases in which the cosmetic result is all that can be desired are successful, meaning, the operator has attained all that he proposed or could do by his operative work. This may not be ideal, but it is successful surgery, and it is not even essential that we have binocular vision in all our operations. In some cases the fusion power may return in time after the deviation of the optic axes is corrected, particularly in the cases of amblyopia ex anopsia.

When my book on strabismus was issued some years ago, I clearly proposed two distinct classes of strabismus, basing the classification on the operative procedure needed in each class. In the first class I placed all those cases of squint that presented well-marked congenital amblyopia, and in the second class those cases of squint which presented good vision in the fixing eye and amblyopia ex anopsia in the squinting eye. That is to say the vision of the squinting eye is reduced, but may be improved by the usual methods after correction of the strabismus.

It is in these cases that we may attain the ideal operative condition of perfect fixation, with true binocular vision. This broad classification may include all cases of squint, though that peculiar condition known as "antipathy to fusion" may sometimes delay the final result. It may be somewhat difficult to draw the line as to when the first class ends and the second class begins, but it has been my past experience that if the vision of the squinting eye is less than 20/200, we do have the visual anomaly of congenital amblyopia. If we find the vision better than 20/100, we have an amblyopia ex anopsia. I have seen the vision decidedly improved in these cases of squint after a suitable operation and the use of glasses. A division like this may be very arbitrary, but many years of observation have convinced me that it is very nearly correct, and that the exceptions may prove the rule.

Now give each of these classes their special operative procedure, and in all we will correct the deviation of the optic axes, and in some bring about the ideal condition of perfect binocular vision. We can correct the deviation of the optic axes in all cases presenting the condition noted in the first class, but in the final result we must not look for or expect any improvement in the visual acuity, but only a permanent correction of a cosmetic defect. And in the second class if we adopt a procedure which I prefer, we will not only correct a deviation but may restore binocular vision. Granting these assertions are correct, it seems to me that we have two propositions: First, we should not have any failures; and second, we should know what is the proper operative procedure. Now let us briefly consider Bielschowsky's two rules: "Operate only when all other means fail." This may be a good rule to follow, but my experience seems to convince me, as I have already stated, that all cases of squint should have some kind of operative interference, but it must be the proper operative procedure and not the usual

method too much in vogue at the present day. For simply to cut the muscles (tenotomy) until the squint is corrected will not do. That procedure too often produces a failure to correct, or a very decided impaired motility or even a divergence. This objection covers his second rule, "An unimpaired motility of the eye must be assured afterwards."

Passing now to the cases of latent squint or heterophoria, we have no loss of fusion force in these. Their vision is perfect in each eye; with binocular fixation and under normal conditions they have no diplopia, and yet we cannot draw the line between fixed and latent squint when examined in a scientific manner. Objectively it may be simple after the eye has turned in permanently, but before, when we see these cases, with no squint in childhood, a history of continued muscular asthenopia and eventually a fixed squint, the abnormal rotation of the eye has overcome the fusion sense and the squint becomes obvious. For some reason the eyes have lost the power to preserve binocular vision, and one eye turns inward, outward or upward. If this be true, it offers an argument in favor of the statement just made, that every case of squint, both latent and fixed, if of sufficient degree, should have the proper operative procedure, even though Bielschowsky asserts that operative treatment is not advisable in latent squint, for "what the near vision gains the distant loses"—a statement that can only refer to a tenotomy.

The object of introducing the subject of heterophoria in this paper is for the purpose of showing the close relation of latent and fixed squint; for an objective squint inward which is fully corrected by glasses as regards the visual lines is still a case of esophoria. In other words, the cause remains, though the cosmetic result may be perfect. But the question as to the future arises. I have just said the esophoria remains, and this child as it takes up the advanced studies of our present school system will have certain symptoms of muscular asthenopia. I will illustrate with a case which is taken from my case book.

Miss K. D., aged 7 years when first examined, 1896. Periodic squint; second class; onset, second year; hypermetropia, 3.50 D.; vision 20/30 each. No squint with refractive correction. Her power to fuse is very low. In 1905, after five years, fusion better. Fusion much better in 1910. Stereoscopic vision. Duction 20 degrees in, 15 degrees out. This case shows a decided tendency to squint with either eye; some loss of fusion and constant change of glasses as the asthenopia returns. The tropometer shows increased power to turn in by right eye. Would not a shortening of right externus correct the inward tendency?

From this case we may say that the hypermetropia and the loss of fusion was the cause of the strabismus, but we cannot base a theory on one case, and the ever-present imbalance of the rotational power is the true cause, as shown by the tropometer. I am fully convinced that if the outward rotation had been much less, that glasses and fusion exercises would not have been of any assistance unless an operation had been performed. Hence in my opinion hypermetropia or fusion defects, *per se*, are contributing causes of squint, but there must be an imbalance of muscular power to rotate the eyes about their respective center, to cause squint, and that the innervation of the muscles is the simple child of a too active brain.

I will illustrate this by two interesting cases, both with good visual power:

Mr. and Mrs. M. G., ages about 38. She had periodic squint since childhood, constant asthenopia. He has fixed squint in right eye, no asthenopia. Rotation: Mrs. G., each eye 55 degrees in (normal), 45 degrees outward (too low); Mr. G., 60 degrees in (too high), 30 degrees outward, right eye, and 35 degrees outward, left eye (very low). As there is less outward rotation in right eye, this eye turns in.

Turning now to the squint of the first class, as I indicated before, I find this illustrative case of convergent squint with right eye turning in, and amblyopia.

D. N., aged 16, onset at third year. Right vision 20/200, no glass; left vision 20/20, Hm. + 1 D. Hyperopia + 3 D. Has worn full correction for some time, squint remains the same. Rotation: right eye 60 degrees in, 20 degrees out; left eye 55 degrees in, 20 degrees out. Here we have a decided tendency for inward rotation, increased in the right eye, which is amblyopic and turns inward. A case giving an examination similar to this with such low outward rotation can only be corrected by a proper operative procedure, and glasses will be useless. I may add that after the operation he had perfect fixation. An exactly similar case, Mrs. M. C., first examined in 1901, has the same refraction and the same amblyopia in L. E., vision 20/200, and yet she has no squint, and good fixation.

Now from this case we cannot say hypermetropia or fusion or amblyopia causes squint, yet all these conditions are present. Why does she not have the same squint as D. H.? Let us look at the rotation. The left eye shows 60 degrees in and 40 degrees out. This should indicate convergence excess. But the right eye shows only 50 degrees in and 55 degrees out, in other words the right eye really shows divergence, and we have no muscular

tendency to deviation and no squint. This case is a true case of dextrophoria, pure and simple. In heterophoria or latent squint you will find the same want of outward rotation, though of less degree, and in exophoria the same deficiency of inward rotation showing and demonstrating the necessity for this examination of the ocular power to rotate the eye, and thus proving the muscular or primal cause of all cases of fixed or latent squint. From my experience along these lines, it seems to me that the true or primal cause present in all cases is shown by the loss of power to rotate the eye, which I would place at 100 per cent.

I would suggest these averages as contributing causes:

Ocular Defect	Per Cent.
Hypermetropia	75
Amblyopia, congenital	50
Loss of fusion.....	10
Antipathy to single vision.....	5
Other refractive errors.....	10

This table is not for actual comparison of cases, but is drawn from an observation of all my cases of fixed and latent squint.

In concluding this argument I must say that this seems to be a plea for the muscular theory of squint as the true determining cause, complicated and advanced by the contributing causes previously mentioned. In support of the theory I will quote from others who have made a very careful study of heterotropia. Duane would divide these cases into two classes: Class A, a congenital deviation. This must mean an abnormal muscular balance, and applying the remedy to the cause, he advocates operation only. Evidently Class A is muscular. But why one without the other? Class B, acquired from causes active after birth. What causes? Amblyopia, hypermetropia, fusion, etc., yet none of these causes will produce squint, *per se*; they must have an abnormal muscular balance, with a weak externus. If, then, it becomes a question of what procedure to adopt on our first examination of a case of squint, let us consider the rotation of each eye about its center. If the excursion of the optic axes shows a reasonable degree of outward tendency in convergent squint, then our atropin, fusion and glasses may be of service, though I regret to say the patient may yet suffer from an esophoria, and not an esotropia. *Per contra*, if we find a weak outward rotation in the same condition, then our cor-

rection of the contributing causes will not correct the squint. We must operate according to the indications of the tropometer, so well stated by Howe of Buffalo—a statement which I shall refer to later. In this connection it has been interesting to me to note that Risley of Philadelphia, in a paper reported in the *Ophthalmic Record*, July, 1912, says that our insufficiencies of the ocular muscles may be divided into two groups—relative and absolute. The former, or relative conditions, which may be due to accommodative action, does not refer to the present paper, but if we study the action of the group called absolute, we find these words: "They depend upon some anatomical fault, some anomaly in one or more of the extra-ocular muscles." No more, no less, and in which we may state that the absolute insufficiencies are due to muscular action. Now in this group if we place all cases of strabismus, and according to Risley, these absolute cases of strabismus are due to muscular action, then this seems to me to be the muscular theory pure and simple. Furthermore, in the same paper Risley states that in many cases the fault may be "an abnormal attachment of the muscle to the anterior segment of the globe." Very true, but is not this an anatomical fault in the muscles themselves, an increased or a decreased power to rotate the eyeball about its center of rotation? Accepting these two anatomical conditions, we must have a change in the power to rotate the eye, a power which can always be fully demonstrated by the study of the findings of the tropometer.

Now we come to the question, what shall we do or how shall we treat these cases of squint? I cannot accept the suggestion of glasses at babyhood, as advised by many writers. This advice to put on glasses when a baby shows a tendency to squint is not fully justified in my opinion, nor do I think it in any way useful. It is true that our experiences in all these cases may differ, as one's foresight is not always correct; but one's individual experience is useful in many ways, and my own observation has convinced me that, as the true cause of squint never changes, we may have the same success with our glasses, our exercises, our atropin, etc., when the child is old enough to make a correct diagnosis of the refraction, as to try these measures before the child has left its mother's breast.

The statement has been made that squint may be due to any number of causes, and so we try all these various suggestions hoping to find the cause, and after a few years of futile effort,

we tell the mother we must operate; and then guess where and what the operation shall be. One tells us tenotomy, but what muscle shall we tenotomize, or go on cutting until we reduce the inward rotation to a low degree? Another says advancement, and again we have the question what and where? Now why all this uncertainty? Cannot the writers tell us where to operate and why? O'Brien said, "Twenty or more years ago, before my studies began, faulty insertion or overaction of the external ocular muscles as the etiological factor in strabismus had great vogue." However I object to this statement, for, during thirty years of active work in ophthalmology, and twenty years in one of the largest eye hospitals of New York, I know that the "muscular theory," so called, was not in vogue in that city. And, furthermore, I want to state that the old theory of Donders' antithesis has always had first place, followed by that of Worth's theory of fusion. But each and all of these theories have so often been proved useless, that I regard them only as a contributing cause. We must go back to an old theory which our present scientific instruments enable us to prove — and that this old theory was correct, is shown by our examination of the rotation of the eyes. If then we may say there is a true cause for squint, a condition which is found to be present in each and every case, I would state it first in the words of Dr. L. Howe of Buffalo: "We should ascertain by means of the tropometer a turning is due to the excessive action of the adductors, or insufficient action of the abductors. On that depends not only the diagnosis, but also the answer to the question whether to make a tenotomy or an advancement. Until we do agree in this uniformity of definition we will continue to 'flounder in confusion.'" I fully agree with this statement, for it seems to prove that the true cause of squint must reside in the muscular balance of the eyes, and it seems also to suggest to the inquiring mind that weak muscular action associated with any of these contributing causes may produce convergence, that may be corrected by glasses, and that excessive muscular action must demand operative interference. Hence a diagnosis of the muscular balance suggests the proper procedure for correcting the contributing cause, or, if necessary, a suitable operation.

Yes, a suitable operation, very true; but how shall we decide this question? Bear this one point always in mind, strabismus of any form — outside of the pathological cases (any form of

paresis)—is due to a deficiency of rotation of the eyeball from purely anatomical causes, and consequently a strengthening operation is always indicated. Landolt strongly advises an advancement, and I am partly in accord with him; but this operation is quite a formidable one in which we so alter the anatomical insertion of the distal end of the muscle that I prefer the well-known operation of shortening the muscle in its long diameter. My own method of doing this operation with the catgut suture has proved very satisfactory, and since I introduced that method in 1895 we have now ten different methods of performing the same operation by as many operators in the United States. This must prove its usefulness. If both externi are deficient in convergent squint, I do a double shortening at once and then use the glasses. If the result is not sufficient to produce good parallel position of the optic axes, I then do a very guarded tenotomy under cocain, until the squinting eye is in its correct position. This procedure applies particularly to squint designated as the second class. In squint of the first class, in which we have a decided amblyopia, and when the tropometer shows a decided rotation inward and a diminished rotation outward, I have always operated on the squinting eye by shortening the externus and completely tenotomizing the internus. This procedure always gives me an excellent cosmetic result. In heterophoria, after a careful examination of the rotation by the tropometer, I have placed the catgut suture in the weak muscle, with continued and very gratifying success.

Let me say in conclusion, with an operation that is perfectly safe and simple, do not let the words "operate as a last resort" influence you in any way. Decide from your examination that an operation is necessary, and you may be confident of success, and not have to consider "our failures in ocular muscle work."

CONCERNING THE USE OF INVISIBLE BIFOCALS
IN THE TREATMENT OF CONVERGENT
STRABISMUS (ESOTROPIA) IN LIT-
TLE CHILDREN

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Esotropia in children under seven years of age is still accounted for in various ways by various authors. Some cling to the Donders doctrine pure and simple, that vicious accommodative habits are entirely responsible for the abnormal convergence of the vertical axes. Others believe with Worth that defects in the fusion faculty or even complete absence of this faculty is the main difficulty, and still others hold that faults in the development or insertion of the extra-ocular muscles is the most frequent cause of esotropia.

Every serious worker at this subject will admit the following postulates: (1) That a certain percentage of all esotropes under 7 years of age lose their convergent strabismus under the influence of a correct refraction, conforming to Donders' "accommodative strabismus"; (2) that a certain percentage of esotropes under 7 years of age are practically emmetropic or even myopic, and this class is not at all explainable on the basis of Donders' hypothesis; (3) that a certain percentage of very young esotropes exhibit congenital paresis or palsy of one or more of the extra-ocular muscles, resulting in the usual clinical picture of esotropia, and these are often overlooked; (4) that a certain very small percentage of very young esotropes are the subjects of faults either in the development or insertions of the extra-ocular muscles leading to the clinical picture of strabismus convergens; (5) that a certain small percentage of young esotropes are the subject of fine ultra-ophthalmoscopic changes in the retina or optic nerve of one eye, which lead to the clinical picture of esotropia.

As^{*}this communication has to do with functional esotropia only, the last three classes (postulates 3, 4 and 5) fall out of consideration, as they are, to our way of thinking, amenable to operation only. Functional strabismus arrays itself in three classes, namely: (a) Pure accommodative strabismus (Donders);

(b) pure afusional strabismus (Worth), in which the fusion faculty is entirely absent—cases that are reasonably rare and frequently come to operation; (c) a combination of a and b, the hybrid type, represented by a weak or imperfect fusion faculty complicated by a high grade of unequal refraction error. This in our judgment is by far the most frequent type of esotropia in both private and hospital work. It is the type that oftentimes taxes us to our uttermost in our efforts to straighten out the visual axes without resort to operation.

If the fusion faculty be only weakly established by the end of the second year of life, it is quite thinkable that a high grade of unequal refraction error may easily prove an obstacle to the firm establishment of the function of binocular stereoscopic vision. During the third year of life little children generally become much interested in picture books and play with small objects. The accommodative apparatus is thus put upon much strain and may become so vicious in its overaction as to disturb the visual apparatus enough to upset or even set aside the fusion faculty. In other words, with a weak fusion faculty the accommodative apparatus of the eye easily falls into vicious habits. Our task then is not only to train the fusion faculty, but so to put the entire accommodative apparatus completely at rest, that the vicious habits it has fallen into will be entirely broken up.

The correction of these vicious states of the accommodative apparatus have been made up to the present time by the use of practically full corrections and the continuous use of atropin in one or both eyes for one to three months after the glasses were put on. About three years ago, while pondering this phase of the strabismus question, it occurred to me that if we could sometimes secure good results in high esophoria (8 to 15 degrees) by adding anywhere from plus one to plus three sphere to the distance correction to be used in near work to suppress accommodative overactivity, it might be worth trying in the functional strabismus of children; it was in fact carrying the idea to its logical sequence.

It was therefore tried in two cases of pure functional strabismus, both the little patients being about 4 years of age. For distance a practically full correction was ordered with a plus two sphere added for the bifocal segment. For the first month after putting on the glasses, atropin (0.5 per cent.) was used in both eyes each morning. The combination of bifocal glasses and atropin in both eyes seems to be the most complete orthoptic

method yet devised for the abolition of habit spasm in the accommodative apparatus.

As it was experimental, these two pairs of glasses (which were prescribed about the same time) were ordered in lenticular bifocals, but they excited so much comment when worn by 4-year-old children, and subjected the parents to such incessant questions from curious and interested persons, that at the parents' request they were made up in invisible bifocals. By this time, however (at the end of about two months), the beneficial effects of the bifocals had become quite strikingly apparent. The atropin was withdrawn and at the end of two more months both children presented approximately parallel visual axes. Both had a deviation of about 30 degrees.

These experiments are now three years old. One child is still wearing an invisible bifocal as follows:

$$\begin{aligned} \text{R. } + 2.00 + 50 \text{ cyl. } 75 \text{ degrees} &= \frac{5}{8} \\ \text{L. } + 3.00 + 1.00 \text{ cyl. } 120 \text{ degrees} &= \frac{5}{6} \end{aligned}$$

To this is added a plus two sphere. The child is now 7 years of age. The other of these two children discontinued the use of the bifocal after two years and for the past year has gotten along nicely with a full correction for all purposes. Since that time, nine other children with pure functional strabismus have been ordered invisible bifocal glasses. In the younger children (2 to 4 years old) + 3.00 sphere was added. In the older ones (4 to 6 years old) a + 2.00 sphere was added. The average deviation in these nine children was 32 degrees which corresponds exactly with an average deviation found by me ten years ago in a "Clinical and Statistical Study of Convergent Strabismus," *New York Medical Journal*, Nov. 5, 1904.

I have been much impressed with the results shown in these eleven cases, in that I think they are superior to those secured by the methods at present in vogue. In this series of eleven cases, eight now have straight visual axes, two are vastly improved, while the remaining one showed very little improvement. In this last case the original deviation was 40; use of a full correction and atropin two months effected 15 degrees of straightening; an invisible bifocal was then used for six months and this produced only 10 degrees more of effect, so that while the cosmetic effect was somewhat bettered there remained 15 degrees of deviation. This child is now 5 years of age and while the parents are apparently quite satisfied with the improve-

ment wrought by these methods (plus fusion training), I do not feel that the method I have suggested has accomplished anything much worth while in this particular case.

In brief, the idea is to set the spastic accommodation apparatus at rest, and we know of no more complete method than the one here offered, namely, 0.5 per cent. atropin solution once a day for two months, with the use of an invisible bifocal, the atropin to be withdrawn at the end of this time. Naturally fusion training should be carried on all the time in different ways (amblyoscope, stereoscope, bar reading, etc.). Picture books, play blocks, alphabets of all sorts are strictly interdicted up to the fifth year. This may seem a hardship to the child, but the end striven for justifies all the time and trouble required. There will always be parents who become impatient of this slower but surer method of straightening crossed eyes. To such parents it may be frankly said that they are in all probability denying their child the possible privilege of binocular vision.

There is one objection to the use of these invisible bifocals in very young esotropes that should be mentioned; that is that the little patients begin at once to dodge the bifocal segment and for the first week or two bob their heads about a good bit. But they soon learn to adjust themselves to the new order of things and whatever good effect the bifocal produces on the deviation will soon follow.

Eleven cases are far too few on which to base any working conclusions. The only claim made is that this proposed addition to our ordinary methods is simple and harmless; and the hope is expressed that it will offer both a logical sequence to our present methods and a reasonable expectation that a still greater number of young esotropes may not only escape the scissors but also be vouchsafed the blessings of full binocular vision.

Since the above was written it has been learned that Dr. Linn Emerson five years ago presented a paper on "The Treatment of Convergent Strabismus," in which he referred to the use of bifocal glasses for the purpose above alluded to. If any name therefore is to attach to this method it should be known as "Emerson's method."

PARTIAL TENOTOMIES BY THE TODD-HARMON METHOD

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Ever since the writer began the practice of ophthalmology he has been particularly interested in cases of heterophoria. He has tried blurring in esophoria, and prisms and exercises in exophoria. Tenotomies, tuckings, and advancements have produced fair and indifferent results. There has always been a feeling that it was rather a gamble as to just what result would be obtained in these cases. Some form of graduated tenotomy has always seemed the least haphazard and the more scientific procedure, but none of the numerous methods advocated has created enthusiasm enough for the writer to try them.

In the *Ophthalmoscope*, January, 1913, Dr. N. Bishop Harmon of London, England, published an article entitled, "Lengthening a Rectus Tendon in Squint Operations." The writer was so much pleased with the simplicity and ease of control in the operation described that he has since, with gratifying results, performed the operation four times on patients with phorias, who had marked general and locally distressing symptoms. The liberty is taken of quoting below a portion of Dr. Harmon's article, to refresh the reader's memory.

"The delicate methods of Stevens, Landolt, and Grimsdale require much patience, and then the result depends on a frail suture. Verhoeff's partial plastic tenotomy is extraordinarily neat on paper, but is difficult to perform on the living subject, and I am not convinced that any real slacking of the tendon is secured. I devised a method of three cuts which gave me all I wanted. It was so simple that it seemed that some one must have done it already, and in Casey A. Wood's new textbooks of eye operations I find nearly the same method figured; but if the author of the operation—Dr. Todd—will forgive me the criticism, it seems that he has just stopped short of doing what he set out to do. The steps of the operation, and the effect, can be seen very well if it be practiced on a piece of narrow adhesive strapping stretched between two fingers of an assistant.

NOTE: Since this paper was written the writer has become convinced that this operation was performed by Dr. F. C. Todd some six years previous to Dr. Harmon's article. Therefore the title has been changed, thus giving Dr. Todd credit for the precedence and Dr. Harmon credit for once more calling it to our minds in a most concise manner.

The tendon to be lengthened is exposed a short distance from the insertion. Supposing it to be an internal rectus, a small vertical buttonhole is cut parallel to the plica semilunaris and close to its edge. Tenon's capsule is cut and pushed back similarly, a squint-hook is passed beneath the tendon and slowly lifted, so as to draw the tendon into view, then two cuts are made in the same edge of the tendon, one on either side of the hook, as far as possible, so as to divide the tension at each cut up to the middle line of the tendon. Then the hook is pushed slightly to one side so the cut may be made from the opposite edge of the tendon, between the two cuts already made, and this cut is made from the edge right across the middle line of the tendon so as to sever *two-thirds of its width*. Immediately the third cut passes beyond the middle line, the tendon will be felt to give and it will yield distinctly as the cut is continued."

Discussions of this operation, and operations performed for the same purpose, elicited the opinion that the immediate result would be no more than ten degrees lessening of the phoria. In spite of the discouraging consensus of opinion, the operation was undertaken with some degree of confidence. In the following case the operation was done on the internal rectus of each eye.

CASE 1.—Nov. 15, 1912. Mrs. H. W. F., myope, aged 34, severe headaches for the past fifteen years. Examined repeatedly with and without a mydriatic. Complains of pain through right eye, with no full relief from pain at any time. Appendix has been removed, turbinates operated on, and an Alexander done. Has been in a sanitarium for nervousness several times. She is often awakened in the morning by severe pain in head. Fundi normal, pupillary reaction good, cornea clear, tension normal; small opacity in posterior surface of lenses. Vision O. D. 20/30 +, vision O. S. 20/70 +. Esophoria 17 degrees at twenty feet, tested with 12-degree prism base down, on the right eye. Esophoria between 12 and 15 degrees at twelve inches. Homatropin ordered for examination.

Nov. 16, 1912. Ordered correcting glass with a 3-degree prism base out, in either eye.

Dec. 13, 1912. Above glass gave relief for four days, when same symptoms came back. Ordered:

O. D.: — 0.50 cyl. ax. 170 with 6 degree prism base out = 20/20.

O. S.: — 1.00 sph. — 1.25 cyl. ax. 10 with 6 degree prism base out = 20/20.

Dec. 31, 1912. Last glasses do very well, though cannot read or sew, because she complains of seeing her nose.

Esophoria 17 degrees plus. Ordered $+0.75$ sph. added to her distance prescription with 6-degree prism base out, O. U.

Jan. 24, 1913. Old symptoms returned again. Partial tenotomy advised on left internal rectus. The operation was done according to Dr. Harmon's method, described above. The middle cut was carried a little farther than two-thirds the width of the muscle, as the greatest possible lengthening was desired. Immediately after the operation prism base down before the right eye showed an esophoria of 2 degrees.

Jan. 29, 1913. There are no headaches at all. Esophoria 3 degrees at twenty feet. No movement with cover test. Ordered distance glasses prescription without prisms.

Feb. 12, 1913. Patient had severe headache again, complains of "pulling" of the right eye from inner canthus. Esophoria 6 degrees at twenty feet. Patient insisted that operation be done on right eye because of the "pull." Right eye was operated on without incident, but the middle cut was not carried over two-thirds the width of the muscle, as only a moderate shortening was desired. The eyes were not tested immediately after this operation because of the patient's nervous condition.

Feb. 22, 1913. Conjunctival stitches were removed; orthophoria at twenty feet.

March 6, 1913. Cover test shows no movement. Feeling as though eye were "held in a vice" no longer present. Eyes white and quiet. Esophoria without glasses $1\frac{1}{2}$ degrees at twenty feet; with glasses $2\frac{1}{2}$ degrees.

Sept. 24, 1913. No movement with cover test, $2\frac{1}{2}$ degrees at twenty feet. Orthophoria thirteen inches.

June 11, 1914. No movement with cover test, with and without glasses. Esophoria without glasses at twenty feet, 2 to $2\frac{1}{2}$ degrees; with glasses 3 degrees. Patient says that she is free from previous headaches and other distressing symptoms.

CASE 2.—July 24, 1912. Mrs. H. M. D., aged 36, has had severe frontal headaches for past month; has been given glasses to rest eyes. Vision: O. D. 20/15 —, O. S. 20/15 —. Esophoria, 12-degree prism base down, twenty feet, 15 degrees; at thirteen inches, 9 degrees. Solution 1 per cent. homatropin ordered for examination. Ordered: O. U. $+0.37$ sph. $+0.25$ cyl. ax. 90 with 4-degree prism base out.

Aug. 3, 1912. Complains of blurring with the new glasses, but advised to continue using them.

Oct. 14, 1912. Esophoria has increased to 20 degrees at twenty feet. Ordered same prescription as above with 8-degree prism base out, O. U.

Nov. 7, 1912. Last glasses cause headache and general discomfort. It was decided to try a marked blurring glass with less prism. Ordered: $+1.25$ sph. $+0.25$ cyl. ax. 90 with 4-degree prism base out, O. U. Esophoria at twenty feet, 26 to 28 degrees.

Nov. 19, 1912. Some trouble wearing glasses; advised to continue their use.

Jan. 28, 1913. Broken the last glasses. Esophoria twenty feet, 27 to 28 degrees. Marked movement with cover test. Operation advised.

Feb. 9, 1913. Operated on left eye, doing partial tenotomy. Middle cut was made as long as possible. There was so much pain resulting from stitching up the conjunctiva that the immediate result of the operation could not be tested.

Feb. 11, 1913. Patient had pain for about four hours after operation but slept well. No headache.

Feb. 14, 1913. Some discomfort because of stitches. Esophoria 12 degrees, twenty feet. To wear old glasses.

Nov. 6, 1913. Esophoria 15 degrees twenty feet.

June 11, 1914. Esophoria at twenty feet, with 12-degree prism base down, 10 degrees; with Maddox rod 14 degrees. Marked movement, cover test. Patient says she will have her other eye operated on next fall.

CASE 3.—April 7, 1913. Mr. W. F., aged 62, marked diplopia. Cover test shows left eye turning up. Had considerable nausea, and much pain in back of his head, running down the left side of his neck. Vision: O. D. 20/30; O. S. 20/20—. With +1.00 O. U., 20/15. Left hyperphoria, 15 degrees. Operation advised.

April 8, 1913. Partial tenotomy done according to Dr. Harmon's method on superior rectus, left eye. Orthophoria immediately after operation.

April 21, 1913. Patient reports gastric symptoms have gone, and no longer any diplopia, except when he looks very high up and to the right. Eight degrees left hyperphoria.

June 12, 1914. Four degrees left hyperphoria. Is not troubled by double vision, except when he looks up and to the right. Still some slight pain at times in left side of his neck. He has been so comfortable that it was necessary to write and ask him to report.

In summing up the foregoing results there are certain factors which should be taken into consideration. Patients with such marked phorias are of a highly strung, neurotic temperament, and it is a question whether this general condition is due to the phoria or vice versa. It is doubtful if it is ever possible to bring out all the latent phoria; however, the effort was made in the first two cases, to bring out all the esophoria present.

In the first case the immediate postoperative result showed a correction of fully 15 degrees. Eleven days after the first operation there were 6 degrees of esophoria and patient still had severe headaches. A few days after the second operation there were $2\frac{1}{2}$ degrees of esophoria at twenty feet, that is, $3\frac{1}{2}$ of the 6 remaining degrees had been corrected. In this second operation the middle cut was carried very slightly beyond the center of the muscle. Fifteen months have elapsed since

the last operation and 3 degrees of esophoria are present; the patient has freedom from the previous distressing symptoms, in spite of the fact that there has been much illness in the rest of her family.

In the second case the postoperative result showed a correction of 15 degrees of esophoria, leaving 12 degrees uncorrected. The patient reports improvement in general condition and less nervousness, and wishes the other eye operated on in the fall. There are still 12 to 14 degrees of esophoria uncorrected.

In Case 3 the immediate result after operation was orthophoria. Fourteen months have elapsed since the operation; there are 4 degrees of hyperphoria present, showing an apparently permanent correction of 11 degrees. The patient has been comparatively comfortable.

Since this report was begun the writer has had occasion to operate on several strabismus cases. In two instances the external rectus was tucked, and the internal rectus was partially tenotomized according to Harmon's method. The procedure proved quite successful and gave a sense of security very foreign to that usually felt after a full tenotomy operation.

DISCUSSION ON PAPERS OF DRs. VALK, REBER AND LOWELL

DR. LINN EMERSON, Orange, N. J.: Let me say that I was one of the earliest men in this country to take up the work of Dr. Worth, and I am an ardent believer in his teachings, always have been and always expect to be. About ten years ago I advocated the use of bifocals in the treatment of these cases described by Dr. Reber. I have treated more than fifty cases with bifocals. The plan of treatment is not applicable, in my opinion, to all cases, and if I have time I will speak of this later. In my paper published in 1906 I mentioned the fact that I used bifocals in these cases when under atropin. I make the statement most emphatically, I have never seen a case of convergent, concomitant squint without absence or deficiency of the fusion faculty. Ten or eleven years ago all the cases that came to our clinic were turned over to me for treatment or operative interference, and I found many who did not have enough squint to justify operation. The strabismus is nothing but a symptom. Of convergent squint, 75 or 80 per cent. come under the category of those that are helped by orthoptic measures (hypermetropia, convergent concomitant squint, etc.). Ninety per cent. of these cases, if you can get them before they are four or five years old and treat them with glasses and orthoptic training, can be cured. There are a certain number of other cases in which there are optical defects and anatomical defects and congenital amblyopia which orthoptic treatment will not do good. You cannot tell until you give them a trial, but if you are going to do good you will get marked improvement in a short time. I wrote considerably of this ten years ago and read a paper before the New York Academy of Medicine. I was young and very sensitive, and when I spoke of using bifocal glasses in small children it was received with smiles, and I discontinued talking about what I was doing,

but I have kept on doing it just the same, and I am still curing my cases. Sometimes I put atropin in the good eye and force the poor eye, and sometimes I use bifocals on one eye and not on the other, when I am treating a case in which I am blurring the good eye with atropin. I then have the bifocal on the good or blurred eye to relieve the tendency to convergence in the effort of accommodation. Sometimes a drop in the good eye once a day and the poor eye twice a week or month, and watch every week and lessen the drop when the amblyopia improves. In some cases vision rose from 20/200 to 20/20 or 20/15. No one of my confrères thought I was honest but misled. After watching a series of one hundred cases and curing most of them it was not any wonder that I sat down disgusted and quit talking. But I am an ardent believer in it and I believe a majority of these cases can be cured.

DR. ROBT. S. LAMB, Washington, D. C.: I wish only to speak favorably of the remarks made by Dr. Emerson. I wished to say these same things and to congratulate Dr. Reber on coming before us again on this subject. I do not use the bifocal but use two pairs of glasses. There is very little convergence while a child is playing around, and I do think there is a disadvantage in a bifocal glass, where children are apt to stumble around and get hurt, because of inability of gauging distances accurately through such glasses while running and playing.

DR. FRANK TODD, Minneapolis: I thank Dr. Lowell for bringing the subject before the meeting and presenting this ingenious scheme for demonstrating the effect. This operation I described before the A. M. A. in 1907 and here is a reprint of that article. I have done the operation a good many times. It is useful in strabismus combined with advancement and is useful for heterophoria. I have never intentionally done a complete tenotomy since I began it. You can get just as much effect by cutting far enough on the sides of the tendon, provided you cut all of the fibers in one location or another, and cut far enough, just as much effect as if you sever the tendon completely from the globe. In heterophoria you can regulate the amount you cut far better than in complete tenotomy. It is my custom in heterophoria in these cases that require this operation, to cut as far as I think safe to secure a certain effect, and then place the patient in front of the perimeter and measure the effect. An overcorrection is of course necessary and the greater the heterophoria the more overeffect required. Time does not admit of discussion of what causes us to determine how much effect is required in various cases, and it is difficult to lay down hard and fast rules. In two cases in my experience I have by accident ruptured a tendon completely and thus gotten a complete tenotomy. In the first I immediately stitched it up, which I advise any one to do. The other case teaches a lesson regarding this operation. I did not suture the tendon but allowed it to go on for several days when I resutured it. This was the case of a girl of 14 who had been under my care for six years. I had kept her fairly comfortable by the various methods up to that time. This last spring she was wearing 5-degree prisms in each eye, base in. I did this operation on one eye and secured an immediate effect of 11 degrees. I did not then operate on the other eye, although I knew it should be done. I never do both at the same sitting, because I do not know what the ultimate effect will be. The effect proved to be six degrees. Then later I operated on the other eye, desiring to secure as much effect as possible. My assistant pulled through the hook and the muscle ruptured. I did not suture it, but a week later I found the eye turned inward and the rotations outward were defective and she was convergent. I picked up with a hook the muscle that had been cut and found there had been no effort at reattachment. I sutured it to its former attach-

ment. Three months later this patient showed three degrees of esophoria for distance, two degrees of exophoria for near, with relief of headaches and ability to carry on her studies. This is exceedingly interesting as a lesson. In Dr. Lowell's hands this operation has apparently proved to be a cure in the case of those patients he has operated on. It does do that in many instances, but it is not a panacea. I have operated on a few patients in whom ultimately a large degree of effect was lost and overcome, and that will occasionally occur. But I do not think there is any tenotomy but that we must occasionally expect such results. This has the advantage of simplicity, and enables the operator to limit the effect.

DR. OLIVER TYDINGS, Chicago: There is much to be said on the subject, but one thing struck my attention when Dr. Valk got up to reply. Nothing has been said touching his work. I want to say that I have read it with great pleasure and followed the work of Emerson and Valk. They have taught me the importance of the intelligent use of the instruments that have been devised for binocular vision, but unless there is one fundamental cause for these troubles I cannot understand a good many cases; and as to that one fundamental cause I am in full harmony with Dr. Valk when he says it is purely muscular.

DR. VALK: It seems to me that the object of my paper has not been mentioned at all.

DR. W. REBER, Philadelphia: I accept everything that Dr. Emerson has said. Dr. Valk and I have argued this question vigorously between ourselves. The difference between heterophoria and true strabismus lies in the fact that in the majority of the latter there is not fusion; there is no stereoscopic binocular vision. I do not believe that in 100 per cent. of all cases of strabismus there is loss of power of rotation. I am ready to defend this statement, as I use the tropometer just as does Dr. Valk. I recall cases of convergent strabismus that show excessive temporal rotation. They will also probably show excessive or defective upward or downward rotation. If Dr. Valk means that there is defective rotation not always temporarily but also up and down then I accept his postulate without reservation. Not one word has been said about the training of the rotations. It is strange that we have not thought of training this faculty. In cases of the type referred to by Dr. Lowell, I would train them. The idea is to have the patient fix on some object straight ahead in the primary position. Then, without moving the head, the eyes are carried as far to the right as possible, then back to the primary position; then, similarly as far to the left as possible and then back to the primary position. This is done in cycles of four counts and they carry the experiment up to a count of twenty morning and night.

In esotropics the best refraction is none too good. Unless they have 20/80 vision (a visual fraction of $\frac{1}{4}$) they cannot read small print. If they have less than that degree of vision there is beginning amblyopia. An eye that takes part in the reading act at any distance is not an amblyopic eye. This is the importance of a visual fraction of $\frac{1}{4}$.

The tropometer alone is not all sufficient in these little people. We need also to study the associated action of the muscles, i. e., the question of the eyes taken together (the associated movements) to ascertain whether there are any congenital palsies or pareses of the ocular muscles present. This method of study of the status of the ocular muscles in strabismus should never be omitted.

DR. FRANCIS VALK (closing discussion): I am glad that Dr. Reber should acknowledge exactly the point of my paper. In every case of

squint there is a weak muscle, and that is the primary cause of trouble. In the case Dr. Todd discussed, he had a weak muscle. The only way you can find that muscle is by the study of the rotation of the eye with the tropometer. Then, to correct an imbalance, place the prism over the weak muscle. The point I wish to bring out is that this weak rotation is present in every case of squint. Dr. Reber says you cannot have a case of convergent squint which, examined by tropometer, shows increased outward rotation. I would ask if he found it in both eyes.

DR. REBER: Yes.

DR. VALK: I have seen cases with increased outward rotation in one eye. You all know some cases are corrected with glasses and some are not. If you choose to work for five years trying glasses and they get better, all right and good, but if you examine carefully and find the weakened muscle and do a simple operation on that muscle which may be followed by good binocular vision, it is much better than to wait till the child gets older and goes to school. I do not operate until the child is 7 or 8 years old. Then I can have a satisfactory examination of the rotation. I put these people off and do not bother with glasses at all in a great many cases, because, as Dr. Emerson says, if they are going to do any good it will show very soon. One question I would bring up, because many are perhaps not familiar with it, and I am glad to say Dr. Reber is slightly inclined to agree with me, that is an interesting series of cases as follows (illustrates). The right eye goes in 40 degrees and outward 60. Naturally you suppose that there you have an exophoria. But when you examine the left eye, you will find that it goes in 60 degrees and outward 40. These are actual cases, and Noyes thirty years ago said he did not know why they show esophoria in the distance and exophoria in the near. If he had examined with the tropometer he would see the fault was in the rotation. The weak internus of the right eye will not hold the visual lines together at the near point. In writing of this condition I unfortunately mixed my Latin and Greek and called it *Dextrophoria*. Some of your patients tell you they read by looking to the right or to the left. They hold the book off to one side. This dextrophoria is the explanation. But the question comes up how to correct this condition. You can operate if you put a prism over one eye with the base in and on the other with the base out. You will correct the tendency to deviation sideways. In all operations on the muscles I much prefer the shortening with the catgut suture, and seldom do a tenotomy.

A RESUME OF THE TRACHOMA BODIES AS THE ETIOLOGICAL FACTOR IN TRA- CHOMA AND IN THE SO-CALLED INCLUSION BLENNORRHEA

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Ever since Halberstaedter and Prowazek¹ published their article in 1907, which dwelt on the subject of cell inclusions, now more or less associated with trachoma, there has been considerable research work done on this subject.

Halberstaedter and Prowazek described their findings as follows: "Staining with Giemsa's method, smears taken from a trachomatous conjunctiva displayed dark-blue, granular inclusions lying in the cytoplasm of the epithelial cells.*

"At first these were small, round or oval, but appeared to develop into less decidedly staining granules containing fine red rod-like points, these latter (i. e., these less decidedly staining granules) increasing in number at the expense and eventual elimination of the original blue masses."

These bodies were called Prowazek bodies. Prowazek regarded the red bodies as the etiological factor of a trachomatous infection, and explained the blue bodies, which he named plastin, as a reaction product of the epithelial cell after invasion by the virus, the red bodies.

As the red bodies increased in number, the blue bodies, or plastin decreased, indicating a triumph of the red bodies over the epithelial cell.

The author sought to establish these findings as evidence not only of a characteristic pathological entity in trachomatous infections, but also as the definite causal factor.

Shortly after this, articles appeared by other observers reporting the presence of typical Prowazek bodies in smears taken from cases of ophthalmia neonatorum of non-gonorrheal origin. Haymann,² in 1910, published notes of their occurrence in four cases of gonorrheal ophthalmia neonatorum and was of

* The illustrations accompanying this article are from sketches made from specimens secured by us from cases of chronic papillary trachoma with acute exacerbations; stained by Giemsa's method.

1. Halberstaedter and Prowazek: *Deutsch. med. Wchnschr.*, xxxii, 1907.

2. Haymann: *Klin. Wchnschr.*, April 11, 1910.

the opinion that the cell inclusions were as much a reaction product to the Neisser gonococcus as to a trachoma virus. In any case, he stated that they were not typical of trachoma alone; but of this more anon.

It is a well-known fact that quite a large percentage of cases of ophthalmia neonatorum are not associated with the gonococcus or with any other recognized organism.

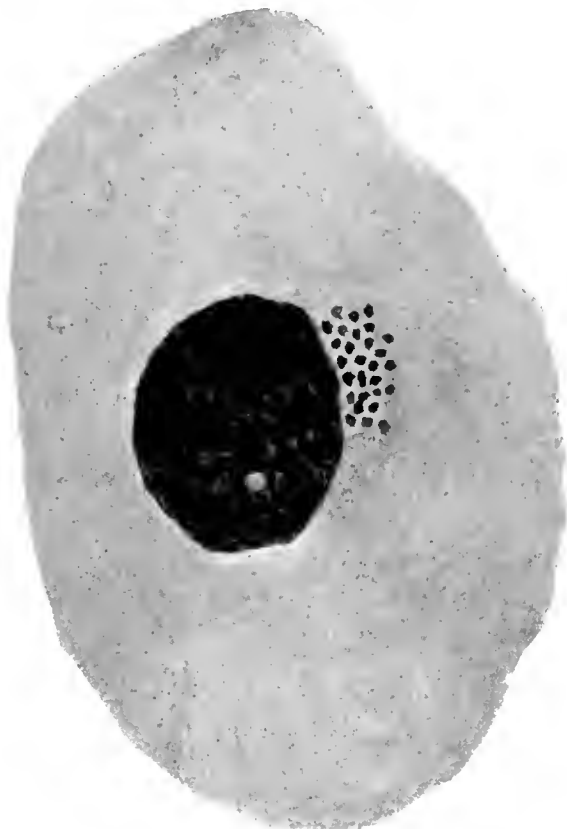


Fig. 1.—Showing an epithelial cell containing the trachoma bodies in an early stage of invasion.

Mr. Sidney Stephenson³ (London), in a recent paper at Oxford, gave as the average percentage of organisms isolated in ophthalmia neonatorum as follows: gonococcus, 65 per cent.; pneumococcus, 10 per cent.; *Bacillus coli communi*, 5 per cent.; other organisms (staphylococcus, Koch-Weeks, etc.), 5 per cent.;

3. Stephenson (not yet published): D. O. Class, Oxford, 1914.

total, 85 per cent.; in 15 per cent. no organisms could be identified.

It is with regard to these cases of ophthalmia neonatorum in which no organisms can be isolated that we are at present interested.

Lindner⁴ and others investigated this type of blennorrhoea and proved to their satisfaction that the typical Prowazek bodies were demonstrable in nearly every case, whereas they failed in

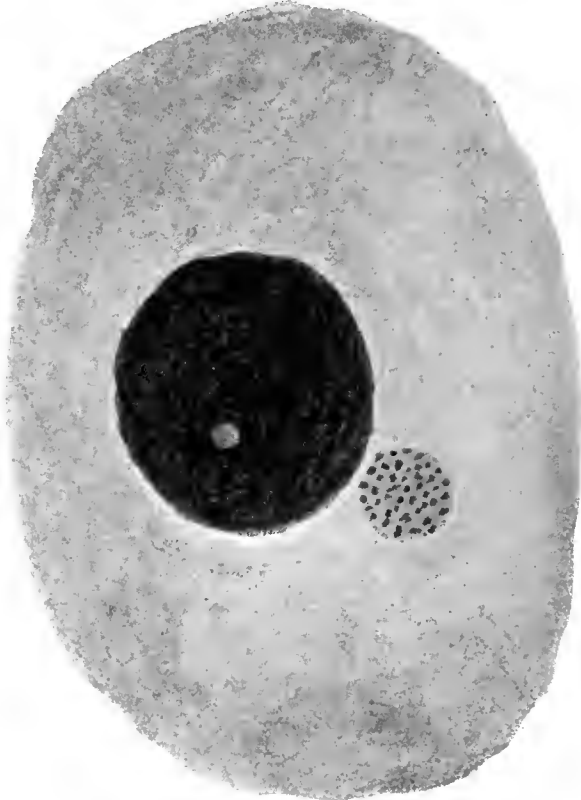


Fig. 2.—Showing a form the trachoma bodies may take. This was called by Prowazek a chlamydozoa.

the great majority of cases to find them in cases of ophthalmia neonatorum in which Neisser's gonococcus could be identified. After this investigation Lindner called this form of infection inclusion blennorrhoea.

Observers also described these cell inclusions in smears taken from chalazia, but more familiarity with the subject of cell

4. Lindner: Arch. Ophth., xxi, 1912.

inclusions showed that these cell inclusions were of a distinct type and could be identified from the cell inclusions occurring in trachoma and in inclusion blennorrhoea. The inclusions in trachoma lie in intimate relation to the nucleus of the epithelial cell and form a distinct compact group of granulations, while those of chalazia are scattered through the cytoplasm and are not in definite relation to the nucleus of the epithelial cells.

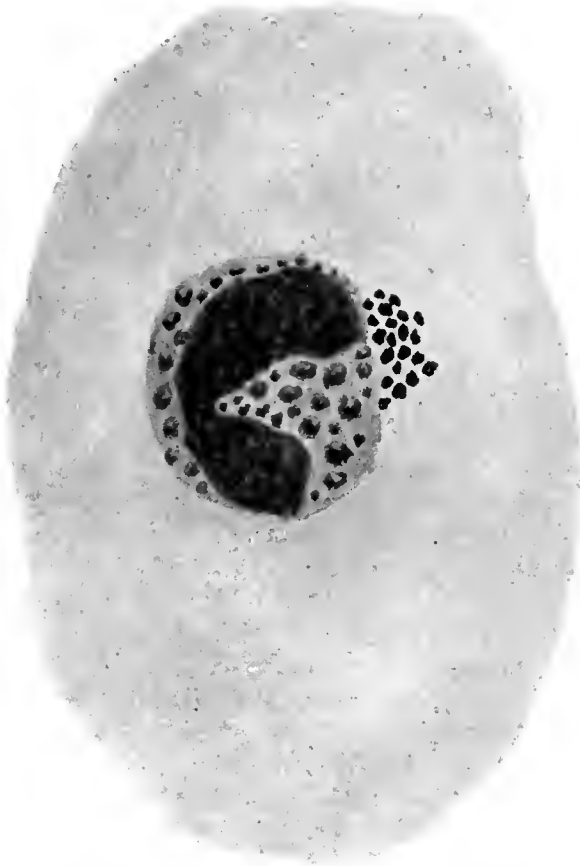


Fig. 3.—Showing a stage where the trachoma bodies have invaded the nucleus and caused its degeneration. This is a late stage.

In 1910 Lindner⁵ convinced himself that the so-called plastin (blue bodies) was but a particular stage of the life history of the virus (red bodies). In his careful preparations he found sharply defined round cocci-like bodies, taking the blue stain, in

5. Lindner: Arch. Ophth., xxi, 1912.

the cytoplasm of the epithelial cells. They occupy an apparent open space or cavity in relation to the nucleus as described above, and on high magnification are found only toward the wall of these cavities. Also, as these blue bodies appear to increase in number, the fine red rod-like bodies make their appearance. The blue bodies, the old named plastin, he termed initial bodies. He found them in an intra- and extracellular distribution.

At this stage, then, it had been established that these bodies occur in trachoma and in non-bacterial blennorrhoea; the question thereupon arose, is the virus one and only one?

Further investigation went to prove that in a number of cases of inclusion blennorrhoea, the cell inclusions could be found in smears taken from the vaginal mucous membrane of the mother and could be found in smears taken from the male urethra of a certain type of non-gonorrhoeal urethritis.

The conjunctiva⁶ of monkeys was inoculated with some of this material obtained from the vagina and urethra. In nearly all cases the investigators reported the appearance of a conjunctivitis similar in reaction to that of trachoma.

And here Lindner calls attention to a very important point. In the inoculation of monkeys with this virus he found the typical trachoma bodies. As monkeys cannot be inoculated with the gonococcus or any organism causing conjunctivitis, except the cell inclusions, this proves the independence of the infection.

Lindner thinks that the inclusions are living organisms and believes that the genital affection in man and woman and inclusion blennorrhoea are identical.

Woburn inoculated two men with inclusion blennorrhoea discharge and produced a genuine trachoma. In Fuchs' Clinic similar inoculations produced a like result. Histological investigations indicated practically similar pathological appearances in trachoma and in inclusion blennorrhoea.

The inclusions have been found associated with gonococci, pneumococci and streptococci in the infant, but this may be a mere association, explained as being a mixed infection. The inclusions have been found in a variety of diseases, including epitheliosis desquamativa, swine pest, and spring catarrh.

To quote Lindner's article, in defense of the above statement, *The Trachoma Question*, *Arch. Ophthalm.*, xxi, 1912: "To this it may be replied that similar organisms are always discovered when a new virus is found, and the trachoma virus is

6. Hereford: *Klin. Monatsbl. f. Augenh.*, March, 1909.

no exception to this general rule. But it is not possible to inoculate monkeys with the inclusions in swine pest and other forms of conjunctivitis, except with the cell inclusions of the so-called inclusion blennorrhea."

Lindner believes that the virus belongs to the class Protozoa, and further states that he agrees with Prowazek in that the red rod-like bodies are alive and the causal agent of trachoma. Prowazek⁷ used the term chlamydozoa (zoa, an animal, chlamy, an outer covering) because the inclusion appeared to be surrounded by a coat.

Herzog⁸ explained the bodies as degenerated gonococci, a species of involution form. Williams regarded them as degenerated forms of the Koch-Weeks bacilli; others held them due to products of degeneration of a still undetected agent or of a parasitic nature but as yet unidentified.

Noguchi⁹ and Cohen¹⁰ have come to view these bodies as a definite pathogenic organism and they agree with the previously mentioned belief of Lindner that the trachoma virus and that of inclusion blennorrhea are identical. They further hold that the occasional occurrence in gonorrheal conjunctivitis was an accidental coincident which could not be explained by the transformation of the gonococcus into trachoma bodies, nor could the two factors operate to produce typical trachoma.

They arrived at this conclusion when they succeeded in growing the organism (the red bodies) outside the body, adopting cultural methods of a complicated nature and similar in some respects to those employed for the growth of the *Spirochaeta pallida*.

The above statements by Noguchi and Cohen, concluded after much work, entirely contradict Haymann's statement that these cell inclusions are found in gonorrheal conjunctivitis. It is true that the gonococcus and trachoma bodies may be present in the same case, operating to produce an acute form of ophthalmia. This may explain in part the source of error and difference by these observers.

DISCUSSION

DR. F. W. ALTER, Toledo, Ohio (closing discussion): There are some points which we would like the privilege to dilate on briefly. First, I wish to call attention to the fact that the trachoma bodies assume a variety

7. Prowazek: Deutsch. med. Wehnschr., xxxii, 1907.

8. Herzog: Ophthalmoscope, Dec. 1, 1910.

9. Noguchi: Arch. Ophth., March, 1914.

10. Noguchi and Cohen: Ophthalmoscope, July, 1914.

of forms. They most often taken on a diffuse form, but there is also a disposition towards a spherical shape.

Second, the trachoma bodies are always in close proximity to the nucleus. This will be found to be constant in character and is of diagnostic and of identification importance.

Third, there is a tendency of the trachoma bodies to surround and invade the nucleus. This occurs in two ways, by a direct frontal attack and also by a sort of flanking movement.

Fourth, there is a gradual elimination of the plastin or blue bodies and a triumph of the red bodies and an invasion of the nucleus and final disintegration and death of the epithelial cell.

Fifth, it is stated that aside from the non-gonorrheal type of ophthalmia neonatorum, namely inclusion blennorrhea, where we find the trachoma bodies, as well as in true trachoma, these cell inclusions are found in a variety of other diseases. I may say in answer and as a reminder that it is not possible to inoculate monkeys with the inclusions found in swine pest and with other forms of conjunctivitis, but we can inoculate them with trachoma bodies. and the cell inclusions of the so-called inclusion blennorrhea.

Sixth, the fact that we find micro-organisms associated with the cell inclusions in inclusion blennorrhea simply means that we have a mixed infection and an acute process superimposed upon the underlying disturbance, trachoma.

Seventh, it may be said in passing that the element of uncertainty which has been displayed relative to the acceptance of the trachoma bodies as the etiological factor in trachoma and in the so-called inclusion blennorrhea has been due in part, at least, to the failure to recognize the possibility of a mixed infection being associated with the trachoma bodies.

SHOULD THE INTRACAPSULAR METHOD OF
CATARACT EXTRACTION BE ADOPTED
BY THE OCULIST OF AMERICA?

OLIVER TYDINGS, M.D.
CHICAGO

The history of the world's progress has ever been one of commonplace achievements until some master mind, meditating on some commonplace fact or work of another, evolves a concept which he executes and thus advances art. A step has been made, a base established from which coming generations start. Such was the work of Columbus, Newton, Watts, Fulton and the host of immortals who have enriched the world by their discoveries. Such was the concept of Daviel while contemplating the work of Petit, who, by a corneal incision, had removed a lens which an accident had left in the anterior chamber.

The chain of thought which led to this brilliant achievement of the surgery of all time is lost, but the achievement stands. It is the greatest monument ever erected by man, not built of stone, but of deeds, enduring, regenerated and renewed afresh in the hearts of all who have been made to see by this beneficent work.

Whether or not Daviel removed the capsule in this first operation designed to seek the lens in its case (Chaton), there is room for question, as the eye was lost by suppuration; or whether he succeeded in his second effort is open to question as he "removed it in pieces." That he did accomplish it in his subsequent work, the world is fully convinced, since Sharp, in his description of his work after the incision is made, says: "After this you press gently with your thumb against the inferior part of the globe of the eye in order to expel the cataract; and the operation is finished according to different circumstances, as in the manner proposed by Mons. Daviel" (*American Encyclopedia of Ophthalmology*). This monumental concept of Daviel's, unquestionably performed many times by Sharp and others in the eighteenth century, was kept alive by subsequent workers, though often thrust aside for procedures which promised better results, until near the middle of the nineteenth century when

Christiaen, the Pagenstechers, and later Mulroney, Mecher Chaud Rai Behager, Mecher Chaud, and Lieut.-Col. Henry Smith established it on a firm foundation from which I believe it is destined to supersede all other methods.

All operators for the relief of cataract are in full accord as to the desirability of removing the capsule with the lens. I know of no text-book which says to the contrary, and the last word on cataract by Dr. Homer E. Smith of Norwich, N. Y., before the Section of Ophthalmology of the American Medical Association, 1914—"Safe and Speedy Extraction of the Immature Cataract and Lens Following Preliminary Capsulotomy" in speaking of the intracapsular method says: "If it could be done with the same proportion of immediate or ultimate success or with no greater operative risk this would be ideal, for it removes in one step every impediment to the clear passage of light into the eye." And then he adds: "Unfortunately the operative risks are appalling and only in hands especially trained under personal tutelage of its one able exponent are these even then brought within reasonable comparison of the time-tried classic method." From such conclusions I humbly beg to dissent. And yet we are informed by Col. Smith that in the Punjab province practically all, at least 90 per cent., of the sixteen or seventeen thousand cataract operations performed last year were done by the intracapsular method; and this, be it remembered, by general surgeons, and not oculists.

The material for this operative technic is plentiful where one has access to a butcher's shop, and the technic is as readily acquired with a pig's or sheep's eye as on the human subject. Indeed, in many respects they are better, better because more abundant. And then one does not feel that weight of personal responsibility to his fellow man, whose eyes are too sacred for experimental work. Your position is different, you are not trying to operate, but to learn how. These are different undertakings—one filled with grave responsibility and fraught with every possible danger to our patient; the other, involving neither responsibility nor danger to anybody. Every one will admit that "the knowing how" should precede the "doing." How many have so erred? I truly believe that just here is where the difficulty lies.

You are familiar with the old operation and it has proved fairly satisfactory in your hands; so was I. Theoretically you knew Smith's technic and attempted to put it into practice without

"the knowing how" and failed; so did I. Where we erred was in trying to attain to a work of art without the training required to master this art.

Few operators in America do enough cataract extractions in a year to acquire this wonderful technic. So the only way for us to develop this art is in the use of pigs' eyes. Here you can practice your incision, lens delivery, delivery with a spoon and all the various steps of the operation. You can do these things as often as you please, using twenty-five or fifty eyes at a sitting and repeat time after time until you have the consciousness that you know; until by actual contact with material you have worked it out; until you have gained that assurance born of conscious ability to do. Until then, we are not in a position to realize that past experience adds but little advantage to this work.

I have talked with the late Dr. Green, with Vail, Fisher, and Austin, all of whom have operated in Smith's clinic, each and all of whom dwelt on the lightness of his grasp, the exceeding ease and lightness with which he does this work. This is one thing to learn and the more you think of it, the more you will marvel at the wonderful art which he has developed.

I gladly pay the homage of my admiration to the master who out of his generosity has afforded so many of my fellow-countrymen the opportunity to learn that art by witnessing and performing operations. So that some of us have been stimulated to acquire this technic and demonstrate that it can be acquired by an American oculist and the operation on the human being be safely performed by one who has never enjoyed the advantage of tutelage at the elbow of that master mind, who, recognizing the advantages of this method had the temerity to adopt it as a routine practice and from the rich field of India so overwhelmed us with statistics of this operation, I blush for my fellow-countrymen who either condemn without trial, or, even worse, a fault many of us have committed, attempt to operate without first mastering this highly developed technic of Col. Smith, which has been so beautifully portrayed by Dr. Vail, another American oculist who has furnished the profession with the most graphic description of this operation which has ever been penned of any operation with which I am familiar, in the history of surgery.

Whether or not the technic, as now developed is the highest which it is capable of, I question. It was modified between the

time of Vail's and Fisher's visits, and again, I am informed, by Austin of Belleville, Ill., who followed Fisher in a few months, before he reached there.

Greene has suggested some modifications, Fisher others, and it will, after the various operators have tried out their concepts of how it should be done, eventually reach that degree of perfection which it should.

As it comes from Smith it is the safest yet devised for the relief of this condition. And while I do not urge on any American oculist working in his limited field to do the intracapsular extraction, I do say it is a duty he owes his patient and the profession to master the details of this "highly technical operation" in doing whatever operation he may elect. And as sure as day follows night will he gravitate to this, the first concept of what a cataract extraction should include. All who have written dwell on the danger of the loss of vitreous.

What operation furnishes absolute immunity from this accident? You and I have more than once met with it while performing the "classic" operation; and more, we have seen it occur while it was being performed by the most ardent supporter of the "classic" method. We have charged it to every cause except the most frequent, viz., a speculum and the operator's faulty technic. It is often proclaimed that the danger of this loss of vitreous is greater in the Smith operation than in the old. If this statement be modified to read, a slight loss is more frequent, it may be true, and yet this is not borne out by the statistics of either Smith or Vail.

Dr. Vail in the article quoted says: "Having kept carefully myself of over five hundred cases done by eight different operators at Jullunder, seven of whom were visitors, the actual number does not exceed 7 per cent. counting all cases. If you eliminate the complicated cases, glaucoma in juveniles and dislocated lens, the number is not over 5 per cent.—his own result even just 2 per cent."

There are means by which a great loss can be avoided, and while it is no part of Smith's technic, yet experience at our institution has abundantly demonstrated the advantages of Fisher's lid retractor while the incision is being made.

We also believe that Fisher's double hook and needle both add additional security to this operation, and both have been tried out and certainly seem to have a place in this work.

In conclusion I will say, first, that the removal of the capsule with the lens is always desirable; second, that the eyes of pigs or sheep with a properly constructed mask are a good substitute for human eyes in the development of this technic; third, that Dr. Vail has given us the most graphic description of cataract extraction extant; fourth, that the Smith-Fisher technic is the safest yet devised for any kind of a cataract extraction, and fifth, that it is a duty which an American oculist owes his profession to study this technic and learn this art.

A CATARACT INCISION LEAVING AN UNDETACHED CONJUNCTIVAL FLAP WITH BRIDGE OF CONJUNCTIVA ON TEMPORAL SIDE

FRANK C. TODD
MINNEAPOLIS

A conjunctival flap is very desirable after cataract extraction, because of the fact that it more readily seals and hence closes the wound more quickly than is the case where the incision is entirely within the cornea. There is less liability to infection and iris prolapse. Indeed, the advantages of a conjunctival flap seem to be so well recognized that most operators have accepted the incision as a routine procedure. While the conjunctival flap is therefore distinctly advantageous, it does not result in exact apposition of the two surfaces, for there occurs considerable retraction of the conjunctiva. Furthermore, if vitreous presents there is nothing to hold it back.

Kalt¹ of Paris secures the advantages of a closed wound by the insertion of a small silk suture into the cornea before making his incision, tying the suture tight after the cataract is extracted. He seems to secure good results with this operation, but it has not been generally adopted, and the idea of a suture in the cornea does not meet with favor for obvious reasons. C. H. Williams and de Mendoza² also used corneal sutures.

In an article on cataract by Huizinga³ of Grand Rapids, Mich., there is described an operation which the writer credits to Schweiger⁴ and Pansier.⁵ This operation consists of making vertical parallel incisions with scissors in the conjunctiva just above the center of the cornea and down to the cornea. The bridge of conjunctiva thereby created is dissected up, but left attached at both ends. Under this bridge is placed a thread so that the bridge may be lifted up during the remainder of the operation. The customary cataract incision is then made and allowed to come out of the sclera just under the bridge. It is claimed that the thread facilitates extraction. Dr. Huizinga commends the operation as having certain advantages, but states

1. Arch. d'opht., xiv, 1894.

2. American Encyclopedia of Ophthalmology, p. 1706.

3. Reprint from the Medical Standard.

4. Arch. Ophth., May, 1898.

5. Ann. d'ocul., Brussels, 1899.

that its advantages are a more difficult and prolonged operation and slightly more reaction afterward.

In August, 1913, I saw de Speville at Rothchild Ophthalmic Hospital in Paris perform an operation which consists in leaving the same narrow bridge of conjunctiva well up on the eyeball over the median line. The bridge was made with the knife after it emerged from the sclera. He credited Vacher of Orleans, France, with having brought the procedure into practice, but stated that it was formerly described by Des Marres. This operation was described also by Robert Scott Lamb in the *Ophthalmic Record*, November, 1913, who evidently independently conceived of the idea, and to whom we are indebted for having brought it to the attention of American ophthalmologists.

After seeing the work of de Speville at the Rothchild Hospital, Paris, I performed some of these operations. It had certain advantages, but also caused some difficulties. It is often difficult to carry the knife far enough up on account of a deep orbit or some other anatomical condition, which prevents proper completion of the incision, especially on the temporal side. But the main difficulty occurs in the delivery of the lens; for if counter-pressure is made outside of the conjunctiva, the instrument holds the conjunctival bridge down, and does not produce the desired gaping of the wound; and it is undesirable to slip the spoon under the bridge lest the eye move during the operation and it becomes caught (if the eye turns upward suddenly, as is so often the case, the instrument may be abruptly forced into the chamber, being held, as it is, under the conjunctival flap). Then too, it will often be found that the flap holds the lens back in the process of delivery, binding down on it. It was for this reason that Schweiger used a thread, with which he lifted the bridge during delivery of the lens.

To obviate the above objections to an undetached conjunctival flap and yet maintain the advantages, I devised the following procedure and have practiced it in thirty-seven operations during the past ten months with very satisfactory results. In four other cases I have attempted it and found it necessary to sever the bridge. It is more difficult of performance in a deep-set eye, but is usually very simple.

The procedure is as follows: After puncture and counter puncture have been made, the handle of the knife is turned downward so that the end of the blade on the nasal side does most of the cutting, though the heel of the knife is allowed to

cut to some extent. As the incision proceeds, the point end of the knife is allowed to do the cutting. The incision is carried well up on the eyeball in the conjunctiva, so that when it is completed, the nasal incision extends well up above the center of the cornea, ending in the median line of the cornea or to the temporal side. The temporal incision is very short, perhaps about twice or thrice the width of the knife blade, in fact just wide enough to permit the incision to come completely through the sclera at or near the corneoscleral junction (Fig. 1). Thus a considerable bridge of conjunctiva is left on the temporal side. Counter pressure may thus be readily made without placing the spoon underneath the conjunctival flap, and delivery is quite simple through the larger (nasal) opening (Fig. 2).

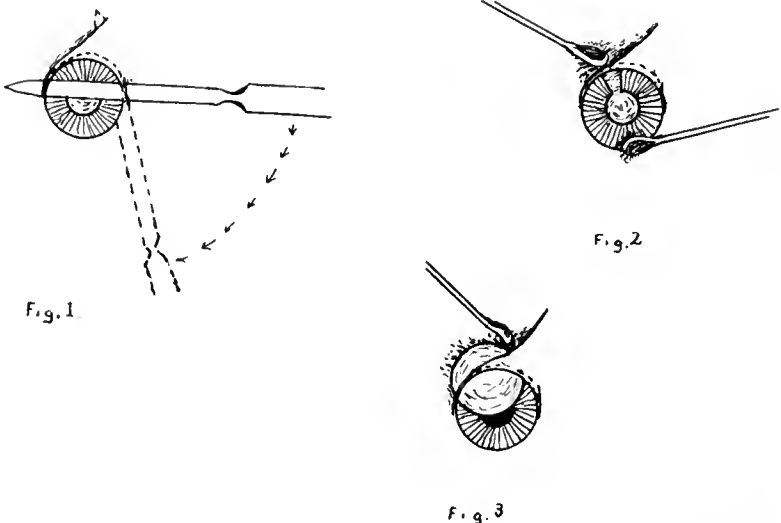


Fig. 1.—Handle of knife on temporal side. Dotted line knife represents final position of knife, which has come through sclera at dotted line and then passed under conjunctiva leaving the latter uncut on the temporal side.

Fig. 2.—Showing how pressure and counter pressure may be made in the usual manner.

Fig. 3.—Showing how the lens may be wheeled out with the spoon if it hesitates under the conjunctival flap.

If it is found that the lens in its delivery hesitates under the flap, the edge of the spoon which has been used for counter pressure may be placed against the edge of the lens and the latter turned out with a wheel motion (Fig. 3). Irrigation may then be freely done through either incision, because the flap is held quite securely and indeed more firmly than when only a narrow median bridge remains. The operator may feel quite free to perform the toilet of the operation to the extent required.

In this operation we have the advantages of the suture without its disadvantages, and the bridge of conjunctiva, which is efficacious in holding the flap in place (as in the Schweiger-Pansier operation or the operation described by Vacher and Lamb). It is an operation which is easier of performance, both in the making of the incision and afterward in the delivery of the lens. In unmanageable patients it is a source of great comfort during the process of the operation and after its performance; and I am sure in several of my cases has prevented the loss of vitreous that might otherwise have taken place.

It is my custom to operate cataracts regardless of their maturity, provided the patient's sight is so reduced as to make him unhappy, and in those cases, where there is cortical matter, free irrigation is practiced, so that all the cortical matter may be washed out. I have found that this operation enables the surgeon freely to irrigate without the danger that exists when a complete incision has been made. Other advantages are the prompt sealing of the wound, thus lessening the liability to sepsis, preventing subsequent prolapse or incarceration and insuring union.

Altogether the operation presents such advantages that I wish to urge its trial on the part of others.

LOSS OF VITREOUS IN THE INTRACAPSULAR CATARACT OPERATION AND ITS PREVENTION

W. A. FISHER, M.D.

CHICAGO

If a cataract operation could be performed without the loss of vitreous, it would make a great step forward and if a loss of vitreous could always be avoided when the lens was removed in its capsule, then the intracapsular operation for senile cataract would be the best operation.

A loss of vitreous during a cataract operation by any of the various methods now in vogue may occur in a certain percentage of operations; and when a loss precedes the exit of the lens, the complication is considered one of great importance. It is admitted by operators who are familiar with all methods of operating that a loss of vitreous occurs more frequent in the intracapsular operation than by the capsulotomy method, and if this loss of vitreous could be avoided, the intracapsular operation could be performed without any more danger to the eye than by other methods.

CAUSE OF LOSS OF VITREOUS

There are two causes for the loss of vitreous: First, the patient may be unruly, but this is not often; and second, the fault is with the operator who does not take all precautions. The patient may be given a sedative sometime before the operation to keep him quiet; but if the operator gives him pain during any part of the operation, he cannot be expected to keep his eye quiet. The greatest responsibility rests on the operator, and he should protect the patient as much as possible against all accidents. The two great factors producing loss of vitreous are pressure on the eyeball by the lids and pressure on the eyeball by the operator in removing the lens. Pressure on the eyeball by the lids can be practically eliminated by discarding all kinds of specula and holding the upper lid away from the eyeball by my retractor and double hook (Fig. 1).

TECHNIC OF THE LID HOOK

To operate on the right eye the assistant stands on the left side of the patient, introducing the lid elevator under the upper

lid, holding it up with his right hand, his fingers resting on the patient's nose and the lower lid is kept down with his left thumb. No. 2.—When operating on the right eye the operator stands behind the patient during the incision and the iridectomy and to the right side and in front of him while extracting the lens. When the incision has been finished and the iridectomy performed, the assistant removes the lid retractor and introduces the double hook. In this position of the lid the operation is finished and the lid closed before removing the instrument, a second assistant or nurse being necessary to hold the eyebrow up during the operation and another to fix the eyeball while making the iridectomy. When operating on the left eye the surgeon makes the incision with his left hand, and the assistant keeps the same position as in operating on the right eye; but if he makes the incision on the left eye with his right hand, standing on the left side and in front of the patient, the assistant should stand on the right side of the patient, holding the lid



Fig. 1.—Author's lid elevator and double hook.

up with the lid elevator in his left hand and keep the lid down with the thumb of his right hand. When the incision is finished the assistant removes the lid elevator, stands on the left side of the patient, introduces the double hook when the iridectomy is made and the operation finished, with the assistant's fingers resting on the side of the face, instead of the nose, and the same position assumed as when operating on the right eye. The operator stands on the right side of the patient in delivering the lens in either eye. There are two positions for the double hook, one for holding the lid up and away from the eyeball, No. 2, the other, the patient being nervous or an impending danger of loss of vitreous, when the lid should be pulled down toward the patient's feet. No. 3.—A little drilling will soon make any physician or nurse a good assistant.

Pressure on the eyeball by the operator in removing the lens need not be so great in the capsulotomy as is required in the intracapsular method and the pressure in the intracapsular method can be greatly modified by the use of my new instrument (Fig. 4). This instrument is a modification of the Smith

spoon, with a Smith spoon on one end and a very sharp needle on the other. The spoon end is used for the delivery of the lens when vitreous has escaped. I do not know of any instrument that will do as well for that purpose. The needle is used to avoid loss of vitreous. The danger of loss of vitreous in the intracapsular operation occurs when the lens appears to be



Fig. 2.—Author's double lid hook, holding the upper lid away from the eye, keeping all pressure from the globe, the assistant's fingers resting on the patient's nose, the thumb of left hand holding the lower lid down, a second assistant holding the brow up.



Fig. 3.—The same as No. 2, except the double hook is pulling the lid forward or down to take off all pressure in impeding or actual loss of vitreous, the double hook being depressed to keep from slipping out from under the lid.

larger than the corneal opening and pressure is exerted that either bursts the capsule or causes loss of vitreous before the lens has been delivered. At this critical moment, when the operator has made what he considers safe pressure and the lens does not move but is sticking in the gaping corneal wound, the point of the needle is stuck into the lens and the lens gently lifted past the obstruction, when the delivery is finished by

pressure on the cornea with the hook according to the Smith method (Figs. 5, 6 and 7).

It would seem that anyone should be able to deliver the lens in its capsule after an experience of 576 operations under the guidance of Colonel Smith. And I am satisfied from this experience that any operator will have an occasional loss of vitreous by using pressure alone, and be compelled at times to deliver the lens with the spoon when loss of vitreous has preceded the delivery of the lens (Fig. 8).

I believe loss of vitreous can be made as infrequent by the



Fig. 4.—Author's needle and Smith spoon.

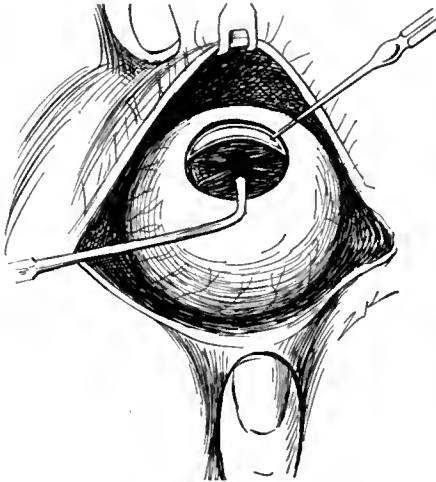


Fig. 5.—Author's lid elevator, assistant holding the upper lid away from the eye with right hand, the lower down with the thumb of the left hand, a second assistant holding the brow up. The hook is laid flat upon the cornea and pressure is made directly back toward the optic nerve. Safe pressure has been made but the lens will not move forward. The needle is inserted into the lens and it is gently lifted past the obstruction and the operation finished by the Smith technic. See figures 6 and 7.

intracapsular method with the Smith technic as occurs in the old operation with the old technic. It is not possible for me to state how many capsules will burst when the needle is used, because it may burst either when excessive pressure is used or when a loss of vitreous occurs. When Dr. Vail of Cincinnati and the late Dr. Greene of Dayton, Ohio, returned from India and explained the Smith technic to the Chicago Ophthalmological Society, they told us to use the spoon to make a little extra pressure when the lens appeared to be sticking in the gaping

corneal wound. Five years later, when I was in India, I found the spoon practically discarded for producing pressure and only used in impending or actual loss of vitreous. Smith made this change because he found that doctors coming to him had great

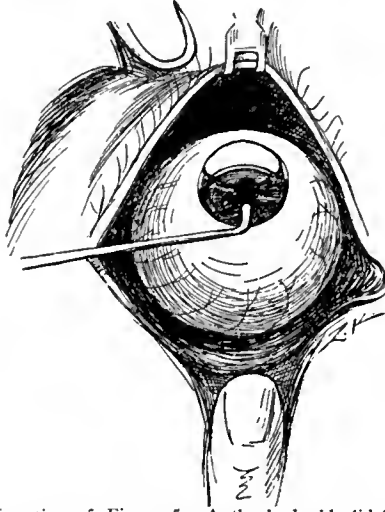


Fig. 6.—A continuation of Figure 5. Author's double lid hook holding the upper lid away from the eyeball with the right hand, the lower down with the thumb of left hand. A second assistant is holding the brow up, while the Smith hook is making pressure directly backward toward the optic nerve. The pressure is kept up until the lens has passed the equator.

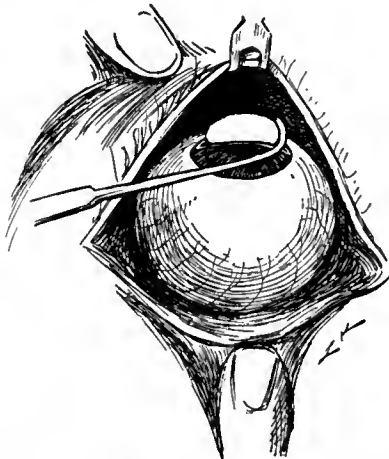


Fig. 7.—A continuation of Figure 6. Author's double lid hook holding the upper lid away from the eye while the Smith hook is removing the lens, which is hanging in the wound. The hook is turned up to avoid rupturing the capsule.

difficulty in using the spoon and that their left hand could not be used as well as the right. He now uses the spoon only in impending or actual loss of vitreous, depending on the hook for

pressure on the lens. I would suggest that the spoon be used only when vitreous has escaped and if the needle is used when there is impending loss of vitreous, the spoon will seldom be used. The more skillful the operator is in removing the lens in its capsule, the less often will he be called on to use the needle or the spoon. The Smith technic should be mastered by good operators on pigs' eyes before an intracapsular operation is attempted.¹ The needle is used only in difficult cases and no more serious accidents are likely to occur than a burst capsule; and in that event the result would be the same as if the old operation had been performed and the capsule left in the eye. If the capsule has been ruptured in a tumbler, that is, a lens

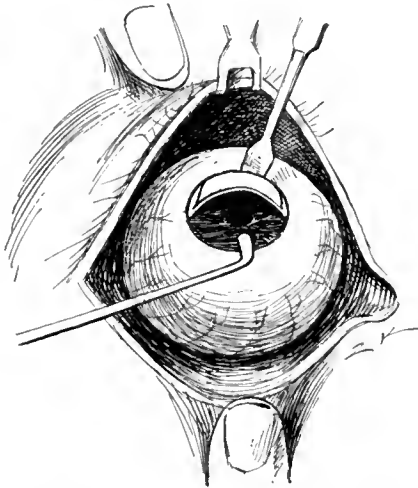


Fig. 8.—Author's double lid hook holding the upper lid away from the eyeball, the lower down with thumb of left hand. A second assistant is holding up the brow. A slight amount of vitreous has escaped; pressure of the hook has been removed and the Smith spoon introduced behind the lens. When the spoon is well in behind the lens, pressure is again made upon the cornea with the hook and the lens is expelled, using the spoon as a background to make pressure. In this manner the minimum amount of vitreous will be lost. This technic can be fairly well mastered by practice upon a dog's or pig's eyes in a mask.

which is delivered lower side first, the zonula will have been broken below and the capsule will be found protruding from the corneal wound, when it can be easily removed with a plain dissecting forceps.¹ If the capsule has been ruptured either by excessive pressure or by the needle while the lens is being delivered in the upright method, as in the immature, the toilet should be made first and then the attempt made to remove the capsule with my capsule forceps (Fig. 9). If the capsule is not removed in this manner, a large opening will have been made

1. Fisher: Ophthalmology, April, 1914.

in it, which will usually obviate the necessity of a secondary operation. If the lids are held away from the globe in a proper manner, a bead of vitreous, but no more, may follow and this need not be considered any special danger.

I hesitate to modify the Smith technic and he may not accept this suggestion, but I believe it will be of great benefit to less skilful operators than Smith. Smith is a very careful observer and will accept reasonable suggestions from anyone and try them out. I offered several suggestions when I was working with him last year and I cannot recall any that he did not try except the needle. I proposed the needle to him when I found the lens sticking in the corneal wound and felt as if the capsule would burst or a loss of vitreous would occur if I made more pressure, but he told me he thought it would be safer for me to stick to the spoon. I did not mention the needle to him again because I was not sure that it would be of any value and, furthermore, I was not in India to teach Smith how to operate.



Fig. 9.—Author's capsule forceps.

As soon as I had arrived in Chicago I had the instrument made and began its use at once and I am using it now in every case when I feel that there is danger of losing vitreous. I have used the needle long enough to convince myself that it is a valuable instrument and I believe it will aid operators in the extraction of the lens in its capsule.

FEW OPERATORS ARE AMBIDEXTROUS

Operators who feel that they cannot depend on their left hand could have an assistant introduce the needle when it is necessary to give this little assistance. The technic of the needle can be obtained by operating on pigs' eyes in a mask. One eye will be quite sufficient for a long practice, because when operating on pigs' eyes it is not necessary to remove the lens but simply to introduce the needle to assist the lens toward the exit then let it slip back into the eye and repeat the technic as many times as is necessary to master it. The spoon delivery can be mastered in the same manner. Operators who do not have competent assistants can perfect them by practice on animals' eyes. I formerly advised the use of the capsule forceps

when the lens could not be delivered by safe pressure, and I am now recommending the use of the same at this critical time.²

We are all striving to avoid loss of vitreous, postoperative inflammation, secondary operation on the capsule and infection, and give our patients the best possible vision. If we open the capsule and extract the lens but leave the capsule in the eye, a foreign body is retained, which has a tendency to cause postoperative inflammation, necessitate secondary operation and increases the danger of infection because we expect and often get postoperative inflammation, which makes it imperative that we open and treat the eye before the corneal wound is closed and thus increase the danger of infection on account of repeatedly opening the eye. If a successful operation has been performed by the old method, the patient is either dismissed at the end of two weeks or more to return for a needling if necessary or is requested to remain in the hospital until this has been done. The patient is usually under observation in or out of the hospital more than twice as long as would be required if the lens had been removed in its capsule; and, when he is discharged and a needling has been performed, we cannot be sure that the capsule will not obstruct his vision at some future time.

DISADVANTAGES OF THE INTRACAPSULAR OPERATION

Loss of vitreous seems to be the one great disadvantage of the intracapsular operation. If this could be overcome I believe the intracapsular operation would be the operation of choice.

ADVANTAGES OF THE INTRACAPSULAR OPERATION

First, the intracapsular operation can be performed as soon as the patient is unable to attend to his ordinary duties.

Second, the intracapsular operation is especially indicated in immature cataract.

Third, the patient is operated on when his health and spirits are good.

Fourth, postoperative inflammation rarely follows the intracapsular operation.

Fifth, secondary operation is not necessary.

Sixth, there is less danger of infection.

Seventh, the average time in the hospital is less than half as long as in the old operation.

Eighth, the visual results are better.

2. Fisher: *Ophthalmology and Oto-Laryngology*, October, 1910.

I have arrived at these conclusions on account of having operated by the capsulotomy method for more than twenty years and having performed more than six hundred intracapsular operations.

DISCUSSION ON PAPERS OF DRS. TYDINGS, TODD AND FISHER

DR. W. A. FISHER, Chicago: It is a great pleasure and privilege to open the discussion of Dr. Tydings' paper and I feel that the members of this Society will be greatly benefited by this contribution. I believe that the members will be encouraged and feel more like looking at the intracapsular operation in a more hopeful manner. If Dr. Tydings can master the Smith technic without going to India and can successfully remove a lens in its capsule, others can learn to do it. Dr. Tydings learned to keep all pressure away from the eyeball by discarding the speculum during every step of the operation and accomplished it by practice upon patients with cocaine eyes before attempting a cataract operation. He also mastered the Smith spoon delivery upon pigs' eyes in a mask. If this technic must be obtained by removing lenses from patients, the operator will soon abandon the intracapsular operation. It is not possible to successfully remove lenses in their capsules if a speculum is used. I am glad Dr. Tydings urges the mastering of the Smith technic and the Smith spoon delivery, which can be done before attempting cataract operation by any method. In *Ophthalmology*, April, 1914, I have described the Smith technic and his spoon delivery. I have also described the technic of keeping pressure away from the eyeball by my lid elevator and double hook.

It seems strange that far-away India is the only place to learn to deliver the lens in its capsule, but in that far-away land there is a man whose equal I have never seen. It seems incredible that in Amritsar, India, more cataract operations are performed in a single day than most of us will do in a year and this is not an occasional day, but the same is done day after day.

While we have no such opportunities here or any other place in the world, we have enough material to make this operation our operation if we choose to do so. I saw Dr. Vail of Cincinnati, who knows the Smith technic and is a very clever operator, operate in Cincinnati and in Chicago. I saw the late Dr. Greene of Dayton, who we all know as a very clever operator, operate in Chicago. I saw him operate four different times, about twenty-five operations, at his home in Dayton, but I could not remove the lens in its capsule without the thought of great danger during the operation. The reason I could not was because I did not go about it in a proper manner. If I had mastered the Smith technic before I went to India in the manner described by Dr. Tydings my trip would have been more valuable.

DR. R. S. LAMB, Washington, D. C.: I was asked to speak on Dr. Todd's paper and will limit my discussion to that. I conceived this idea of cataract extraction with a bridge flap in order to get plenty of room and at the same time keep the circulation. After Dr. Todd suggested the name of Vacher in connection with the operation I looked up the subject and found that Denis and Vacher had written on operations concerning the eye, nose and throat, that this incision had been discovered by accident and they had kept it up by design, realizing the advantage to the patient of a nervous type, the better sense of security on the part of the patient as well as the operator, and freedom from loss of vitreous; the opportunity to close the wound immediately, the better healing and the less liability of infection, in view of the better healing. I could under-

stand their argument, which was the same as my own for doing the operation. Healing took place in about twenty-four hours, and many times after healing it was difficult to find the scar, although it was known the case had been operated on. There is, of course, evident the iridectomy and the absence of the lens. I do all of my extractions with the bridge, however, not quite as Dr. Todd has done it. There seemed to me dangers in his operation, the chief of which is in rolling the eye out; if it should so happen that the patient should do this after the incision, there is a tendency for the wound to gape toward the nasal side of the limbus which would result in loss of vitreous from lack of support at that point. There is an advantage, however, in the facility with which the operation is done. We have all lost aqueous before completing the incision. With this incision, a sweep of the knife, the edge of which can be turned partly toward the cornea, will prevent the iris from being engaged. It seems to me, however, that the incision cannot be made by one who has not done a good many operations. I should say the average operator who uses the seesaw motion in his operation would do better with the flap vertical, rather than toward the temporal side. Above the limbus it is easy to obtain from 8 to 10 mm. of conjunctival bridge. The iridectomy can be easily done by shoving aside the flap at bridge, with forceps, extending the points under the bridge and cornea and grasping the iris and cutting off beneath the flap whatever sized piece of iris you desire. The conjunctival bridge, which cannot be completed with the knife, can easily be completed with the scissors by an extension and tunneling the bridge. As for getting the lens out I may say that it has seemed to me to be better done by a loop and slight counter pressure. I introduce beneath the bridge a little loop, with which you are all acquainted, and tease the upper edge of the lens a bit, at the same time making counter pressure. If the lens does not present quickly I take the loop and go beneath the lens, pushing the lens forward toward the cornea and the bridge and so withdraw it. One great advantage of the bridge is that the operation may be stopped at any stage and you feel perfectly secure in releasing the eye for as much as several minutes at a time, while the patient gets quiet and a little more anesthetic is used; then you are able to proceed with the operation. I believe the fact that the bridge acts as a guy rope in pulling the cornea back into place gives the patient a better sense of security.

DR. HEMAN BROWN, Chicago: Dr. Fisher's paper is a clear presentation of a very important subject—important in any method of lens extraction, but at this time especially, when the intracapsular operation is being more widely discussed and practiced.

Personally I feel confident that this operation will gain more universal favor with added experience, and the objection offered to it, the loss of vitreous, which is too common in all lens extractions, can be controlled and largely prevented by a delicate technic. To this end there are two or three points which experience and observation have taught me should be emphasized. The first is the perfect control of the eyeball; pressure of the lids upon the ball during the escape of the lens is most certain to defeat the end. Immunity from pressure can best be attained by the use of the lid elevator on the upper lid with the brow elevated, and the thumb to the lower lid; in this experienced assistants are all important. The corneal section must be free. Too small an incision can easily abort your purpose.

Another point to which I call your attention is the method of pressure upon the cornea by the hook. The pressure should be directed backward (the eyeball fixed upward), for this is a critical moment in the operation. If the lens is slow to lift with due pressure exerted, the operator is too often inclined to change the angle of his pressure from the pole of the ball,

where pressure is exerted equally on the entire vitreous, to a slightly upward direction, toward the corneal incision, with the result that he directs too much pressure toward the upper and anterior portion of the vitreous, and thus favors a rupture of the zonula and possibly the lens capsule, which might have been avoided by even greater pressure, if necessary, directed in the proper manner toward the optic disk.

The timely use of the spoon or needle will greatly lessen vitreous loss, and no operator should attempt this operation without familiarity with their use.

The success of the intracapsular operation, like that of any other delicate procedure, largely depends upon strict observance of details. It is not practical in all cases—but what single operation is universal in its application? No operator should condemn the operation upon theoretical grounds, much less should the inexperienced attempt its performance, though it is entirely feasible in the hands of experienced operators. In this statistics will bear me out.

DR. MILLETTE, Dayton, Ohio: It has not been my privilege to study this operation at the elbow of the great master, but I have had some experience as an assistant and an operator under Dr. Greene. Personally I am a lover of the operation and think it ideal in about 75 or 80 per cent. of the cases. Perhaps the others are just as good cases to operate on, but I do not feel sure of the patient. The condition of the patient and his tractability, one's control of him and his control of himself, mean much as to the outcome. The incision being very large, there is more danger if he be intractable.

Loss of vitreous is not the bugbear it formerly was. I have seen a third of the vitreous lost and yet very good vision follow. I have also seen a third of the vitreous lost and in two cases intra-ocular hemorrhage followed. I consider the intracapsular extraction of the lens a more dangerous procedure than the older capsulotomy method because of the larger incision and because of the lack of support after the capsule is removed. There is a very close relationship between the posterior capsule of the lens and the hyaloid membrane of the vitreous. I have demonstrated in a number of cases with pigs' eyes and calves' eyes this extremely close relationship by being able to lift the entire vitreous body out of the globe by the lens, the anterior portion of the globe having been removed.

Dr. Fisher speaks of the loss of vitreous being due to the principal features, the lack of skill of the operator—and that is a serious source of loss—and the pressure of the lids. I have seldom seen loss from pressure of the lids, but I have seen most vitreous loss from a third cause, namely, the contraction of the inferior rectus by the patient himself, involuntarily. I have assisted in almost all of the operations of Dr. Greene and have done quite a few myself, and I have observed that the sudden contraction, even slight, of the inferior rectus muscle is the most frequent cause of loss of vitreous. To the man who is qualified the intracapsular extraction of the cataractous lens is the ideal method.

DR. F. H. VERHOEFF, Boston: In connecton with Dr. Todd's paper, my experience with Van Lint's sliding-flap operation may be of interest. Van Lint claims that his procedure guards against iris prolapse and infection. On theoretical grounds it appealed to me so strongly that I determined to give it a thorough trial, and in 1913 the method was used by myself or assistants in all cataract operations on my service at the Massachusetts Charitable Eye and Ear Infirmary. I did not find, as might be supposed, that the added manipulation was disquieting to the patient, but on the contrary that it prepared him for the more serious steps in the extraction. I also did not find that the increased conjunctival reaction

following the operation was of any consequence. Infection occurred in only one case, following an operation by an inexperienced assistant, so that the method cannot be said to favor infection at least. On the other hand, our results seemed to refute Van Lint's contention that the flap tended to close the wound and thus guard against iris prolapse. For we found that too often the anterior chamber was not established for forty-eight hours or longer. In other words, we found that if the flap was sutured under sufficient tension to exert any appreciable pressure on the cornea, it prevented perfect apposition of the wound lips. Many times it was impossible, owing to the character of the conjunctiva, to prepare a flap that did not exert undue pressure and in these cases closure of the wound was always delayed. Simple extraction was performed in three cases, in two of which there was prolapse of the iris beneath the flap within twenty-four hours. In both of these cases the flap was removed, the iris excised, and the flap replaced, the ultimate results being good.

With this experience it was not deemed justifiable to attempt further simple extractions by this method. In one case in which there was loss of vitreous, the escaping vitreous was held under the flap, causing the cataract incision to gape. In several other cases of vitreous loss, however, this did not occur. Owing to its tendency to prevent apposition of the wound surfaces, I have now abandoned the operation as a routine procedure.

DR. OLIVER TYDINGS, Chicago: I would say there are but two justifiable reasons for the loss of vitreous. Dr. Fisher names the unruly patient. One is hardly justified in operating on a patient while he is unruly. One of the best operations Dr. Fisher ever did was upon a patient of this kind. I profited by it and believe he did also. He took that man into his confidence and explained how easy it would be to lose the eye, quieted his fears, and did a beautiful piece of work and got a perfect result. So I would eliminate that cause and add another—the use of a speculum while making an incision. This is a grave fault where the zonule is weak. It was recognized by Dr. Fisher many years ago and avoided by both of us since.

As I stated in my paper, we believe the double hook of Fisher has some advantage over the single hook of Smith. I have used it and found it satisfactory. The needle which he added to Smith's spoon I believe has a place in this work, and while I have not had occasion to use it, I have seen it used by him several times, and though he failed to deliver the capsule with it in some of these cases, the condition of the patient was better, as the entire contents were emptied, which is not always possible by the classic method.

We cannot dwell at too great length upon the Smith-Fisher technic. The time of operation is when the patient is in the best possible condition, and not worried from needless waiting; the comparative freedom from all postoperative inflammatory conditions is a great boon. The elimination of needless interference after the operation, so long recognized as a surgical necessity in all the other branches of surgery, serves to stamp this the safest and best operation yet designed.

DR. TODD (closing discussion): Several members have spoken to me quietly here about this operation, and it makes me think they did not thoroughly understand it. These drawings cannot well be seen. The operation is not more difficult than the ordinary operation. The knife is passed through the cornea and the incision carried through, and after passing through the sclera it simply continues on and up in the conjunctiva. The conjunctiva being undermined here (illustrates). Adrenalin is used and I irrigate with saline solution, if necessary, before I do my iridectomy. Then the iridectomy is not done in the middle but on the side where

most needed. The location of the incision and flap facilitates the pressure and the extraction of the lens, and I would prefer to do that than to introduce the loop into the wound under the lens, as Dr. Lamb suggested. It avoids the necessity of doing that. While it may be true, as Dr. Suker says, that you do not get less astigmatism with the conjunctival flap, where you go through and bring your knife completely out, it is less when the flap remains undetached. As Dr. Lamb said, it is difficult after healing occurs to see where the incision was. There is a protection against infection, as healing takes place so promptly. In these cases where I do an iridectomy, I do not dress the eye until several days after the operation and the bandage may be removed early because the anterior chamber is restored.

The reason healing is so prompt is because the edges are held in such correct apposition, but mainly because the conjunctival vessels are not cut off and thus nutrition is not much disturbed.

DR. FISHER (closing discussion): When a loss of vitreous occurs it does so at the time of the operation and is usually caused by pressure upon the globe by the lids or pressure by the operator in removing the lens. I fully agree with Dr. Suker that a conjunctival flap will not prevent loss of vitreous. I have not had any experience with the method described by Dr. Todd, but I hope it will be one more step forward in making the intracapsular operation practical. The one great objection to the intracapsular operation seems to be vitreous loss, and if a conjunctival flap is made in the manner described by Dr. Todd and the lens appears to be larger than the opening, I cannot see any objection to my needle being used to aid in the delivery of the lens and in this manner prevent loss of vitreous. I am gratified to know that Dr. Tydings has mastered the Smith technic and has courage enough to say so. If he can learn it others can do the same.

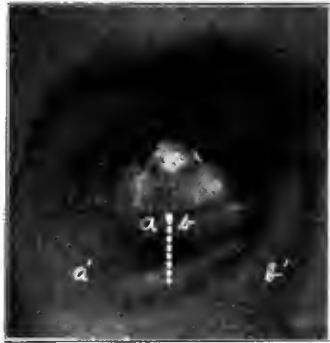
AN OPERATION FOR THE PREVENTION OF SYMBLEPHARON

ELMER G. STARR, M.D.
BUFFALO

The operation herein described is the only one which in my experience has been satisfactory in preventing adhesion between the eyeball and eyelid, following destructive injury to the conjunctiva, especially injuries from corrosive chemicals and involving the fornix.

For obvious reasons the conjunctiva of the lower lid and lower portion of the eyeball are most frequently injured in this way.

The operation consists in completely dividing the lower eyelid from its margin well down to the lowest portion of the cul-



The dotted line indicates cut through lid; the flaps are stitched, *a* to *a'* *b* to *b'*

desac, and stitching the two flaps or halves thus made, one to the side of the nose, the other to the cheek, where they are kept covered with sterile vaselin dressings until new mucous membrane has formed over the injured surface, when the cut edges of the lid are freshened and stitched together in their normal position.

The result in the majority of cases is complete restoration of the fornix and unimpaired mobility of the eyeball.

The accompanying photograph is of a man's eye on which I did this operation more than twenty years ago, following an

extensive burn with caustic lime, involving the lower culdesac. The eyeball is freely movable, as is indicated by the photograph, recently made, and although the cornea is opaque, the eye is free from irritation.

This operation makes it possible to prevent the formation of adhesions between eyeball and eyelid; it facilitates dressing the injured surfaces and by keeping infectious discharges away from the cornea it materially enhances the prospect of preserving this important structure.

SCLEROCORNEAL TREPHINING

ERASTUS EUGENE HOLT, JR., A.B., M.D.
PORTLAND, ME.

All who heard or read the oration of Lieut.-Col. Elliot delivered before this Academy last year, and the illuminating presentation of the subject with which he closed the discussion, realize that it contains much of what he has published in his book or discussed at the various clinics he has held in America. Since he has done so much to help us to cope with one of the most dreaded diseases in ophthalmology, we certainly are under a great obligation to him and should comply with the request which he made at the close of that memorable discussion on trephining the cornea and sclera when he said, "Publish your findings, your failures as well as your successes."

It is with feelings of great obligation to Lieut.-Col. Elliot that I venture to comply with his request and publish the cases of sclerocorneal trephining that I have made since the fall of 1911.

At the Clinical Surgeons' Congress of North America, held in Philadelphia, I saw Dr. L. Webster Fox perform this operation at the Medico-Chirurgical Hospital. The operation appealed to me at once as a great advance in meeting the indications in glaucomatous eyes. At first I performed the operation as described by Col. Elliot, but the majority of my operations have been performed by employing the sliding flap, as practiced by Dr. Fox. I feel, however, that the reasons given by Col. Elliot in regard to the action of the epithelium of the cornea contributing toward the closing of the sclerocorneal opening in the sliding-flap operation are sound and should be heeded provided further experience shows that this occurs more frequently than when the flap is made by splitting the cornea. So far as I have been able to ascertain in my own cases, the sclerocorneal opening in the sliding-flap operations has not been closed. The trephining has fulfilled the objects for which it was made as far as could be expected, as evidenced by the relief of the symptoms of the patients upon whom it was performed. I realize, however, that the sliding-flap operation is open to objection on the ground, theoretically, that infection is more liable to take place than in the operation as practiced by Col. Elliot. When,

however, the conjunctiva is buttonholed, as not infrequently occurs, the sclerocorneal opening is not protected against its being infected or closed by the action of the epithelium as well as in the sliding-flap operation. I have had one infection in twenty-nine operations (Case 18).

I had the pleasure of seeing Col. Elliot perform this operation at the clinic held at the Medico-Chirurgical Hospital by Dr. Fox last year and witnessed the difficulties he had in making the flap. If the sliding flap gives as good results as when the operation is performed according to the original method as devised by Col. Elliot, it must supersede that method in the practice of a large number of operators, as it must be admitted it is not so difficult to perform.

I have used the Von Hippel trephine in all of my cases with so much satisfaction that I can see no reason for changing this practice.

HEMORRHAGE

This occurred in six cases as follows: Case 2, subhyaloid in left eye in the macula region, observed two days after the operation; Case 4, anterior chamber two-thirds filled on the following day; Case 7, profuse expulsive in left eye; Case 12 profuse expulsive in right eye; Case 15, profuse expulsive in left eye; Case 17, intra-ocular. The profuse expulsive hemorrhagic cases No. 7, 12 and 15, were enucleated. These were cases in which it would be good surgery to practice enucleation at once, but as this operation was refused at first, the trephining was resorted to with the hope of relieving the condition and of retaining the eyeball. This had been accomplished in other cases with apparently no more prospects of its being done in them than in those that failed. If sclerocorneal trephining will enable a majority of patients who have painful glaucomatous eyes to retain them comfortably for years after the operation, it certainly is a great boon to them, and for this one use alone it is entitled to a high place in surgical procedures for the relief of these unfortunate persons.

TABLE 1.—SHOWING EFFECT OF IRIDECTOMY ON IRITIS

Character of Iridectomy	With Iritis		Without Iritis	
	No. Cases	Per Cent.	No. Cases	Per Cent.
Complete—				
Case (2) O.D. (5), O.S. (17) and (19)	0	0	4	100
Partial—				
Case (1) O.S. (3) O.S. (7) O.D. (8)				
O.D. and O.S. (9), (10), (11), (13)				
O.D. and O.S. (14), (16) O.D. and				
O.S. (18) O.D. and O.S. (20).....	5	31.25	11	68.75
Without iridectomy—				
(1) O.D., (2) O.S. (4), (5) O.D. (6)				
and (21)	3	50	3	50

The inference to be drawn from Table 1 is that complete iridectomy lessens or prevents iritis; that the frequency with which iritis occurs with partial iridectomy is in proportion of 5 to 16, and without iridectomy 3 to 6.

A 1 per cent. solution of atropin was instilled into each eye at the time of the operation, except in Case 17, which had intra-ocular hemorrhage and the tension remained high. It was repeated every day from one to three times, according to whether the reaction appeared to be normal or excessive.

Experience teaches me that this practice is quite as important after this operation for glaucoma as after the operation for the extraction of cataract, to prevent the occurrence of inflammation of the iris.

CASE 1.—Mrs. S. F. D., aged 40, seen on Jan. 15, 1912. Three days previously noticed redness of the left eye and swelling of the lids with but little pain. Home remedies were tried but the eye grew steadily worse, so that she called her physician who advised her to see me. Patient rather poorly nourished and suffering with excruciating pains referred to the left side of her head. The left eyelids were tremendously swollen and discolored, between which protruded the greatly chemotic conjunctiva. The eyeball felt hard through the swollen lids. Upon separating the lids the steamy cornea prevented a view of the interior of the eye which did not perceive any light. General blood pressure 115 systole. Two years previously this eye had been struck with a ball thrown at a distance of ten feet. Since then vision had been poor. Antiglaucomatous treatment gave only partial relief, so on the next day an Elliot operation was proposed but was rejected. In spite of continued energetic treatment, the eye did not respond and the tension remained high, ranging from 2 to 3. At the end of six days she finally consented to the operation, but at this time an enucleation would have been better surgery. January 21, Elliot operation, with a partial iridectomy, reduced the tension to normal. Recovery prolonged by a severe iritis. Ultimate result good as regards tension, 12 mm. and vision regained light perception. The right eye was not involved, and vision was 1. February 26, returned, stating that two days previously the right eye began to pain her. Vision was reduced from 1 to 0.3+, T. = +2. Under treatment the tension remained about the same. At the end of two days she consented to an operation. Elliot operation without iridectomy lowered tension to normal. This was followed by a mild iritis with an exudate in the central area of the pupil, which has cleared to a large extent so that vision = 0.1+. At the last examination, July 10, 1913, vision had improved to 0.2, tension 15 mm.

TABLE 2.—SUMMARY OF HISTORY OF 21 CASES OF SCLEROCORNEAL TREPHINING

Case No.	Eye	Date	Type	Vision		Tension		Result	Remarks
				Before Operation	After Operation	Before mm.	Date	After mm.	
1	O. S.	Jan. 1912	Acute	Nil	-Pl	+3	June 1914	12	Good
2	O. D.	Feb. 1912	Acute	0.3+	0.2	+2	June 1914	15	Poor
	O. D.	Feb. 1912	Chronic	F., 1 meter	0.8+	+3	April 1912	14	Good
3	O. S.	Feb. 1912	Acute	0.8	0.1	+1	April 1912	15	Poor
	O. S.	Feb. 1912	Absolute	Nil	Nil	90	April 1912	19	Good
4	O. S.	Feb. 1912	Acute	Pl.	0.2-	70	June 1914	15	Good
5	O. S.	Feb. 1912	Chronic	F., 10 ft.	F., 10 ft.	80	June 1914	15	Good
	O. D.	Feb. 1912	Acute	0.5	0.1+	45	June 1914	12	Poor
6	O. S.	Sept. 1912	Absolute	Nil	Nil	90	Jan. 1914	15	Good
7	O. D.	Nov. 1912	Acute	Pl.	0.3	75	July 1914	18	Good
	O. S.	Jan. 1912	Absolute	Nil	65	Failure
8	O. D.	Feb. 1912	Chronic	Pl.	0.1	75	Jan. 1913	18	Good
	O. S.	Jan. 1913	Acute	?	0.1	65	Jan. 1914	u	Good
9	O. D.	Mar. 1912	Hemorrhagic	F., 3 ft.	F., 3 ft.	65	June 1913	25	Good
10	O. S.	Dec. 1912	Chronic	Pl.	0.05	68	June 1913	20	Good
11	O. D.	Jan. 1913	Traumatic	Pl.	Pl.	48	Dec. 1913	18	Good
12	O. D.	Jan. 1913	Secondary Hemorrhagic	Nil	98	Failure
13	O. D.	April 1913	Simple	F., 4 ft.	F., 15 ft.	50	Jan. 1913	15	Good
14	O. S.	April 1913	Simple	Nil	Pl.	60	Feb. 1914	18	Good
15	O. S.	April 1913	Chronic	Pl.	Pl.	55	20	Good
16	O. S.	Aug. 1913	Absolute	Nil	Nil	90	Failure
17	O. D.	Aug. 1913	Absolute	Nil	F., 6 ft.	85	Sept. 1913	17	Good
18	O. D.	Oct. 1913	Chronic	Pl.	F., 14 ft.	78	Sept. 1913	u	Good
19	O. D.	Dec. 1913	Absolute	Nil	Nil	80	Jan. 1913	30	Good
	O. D.	Jan. 1914	Simple	F., 10 ft.	0.2+	35	April 1914	12	Good
	O. D.	Jan. 1914	Iridocyclitis, Secondary	F., 2 ft.	Nil	45	Failure
	O. D.	Jan. 1914	Absolute	Nil	Nil	70	June 1914	45	Poor
20	O. S.	April 1914	Chronic	Pl.	0.5	85	June 1914	15	Good
21	O. S.	Mar. 1914	Absolute	Nil	Nil	98	July 1914	20	Good

Subhyaloid hemorrhage
Later, eye removed on account of an injury.
Immediate result better, V = 0.3—, now lens
hazy.

Immediate result good, 2.3 months after
operation; mild iritis.

Enucleated.
Accurate tests impossible on account of her
mental derangement.

Enucleated.

Enucleated.

Secondary infection.

CASE 2.—Mrs. W. M., aged 59, seen Feb. 1, 1912, with the history that following an attack of la grippe two years previously the vision of the right eye became dim. For some time previous to this sickness a halo around a light had been seen. These spells increased in frequency and duration up to four months ago, when she was awakened early in the morning with excruciating pain in the right eye and the right side of her head. Upon making an effort to open her lids the patient stated that she was unable to see the flame of her kerosene lamp. Heat was immediately applied, and together with a sitting position, much relief was experienced. In a few days the pain lessened, the swelling subsided and the sensitiveness to light disappeared largely, but vision did not noticeably change. Living in a distant town, and with the hope that the vision would improve, as it had in the past, she deferred seeking advice until the present time. Examination of the right eye revealed large scleral veins; hazy cornea insensitive to touch; astigmatism 2.5 D. 90 degrees (ophthalmometer reading); very shallow anterior chamber; pupil widely dilated and immobile; vitreous not clear, so that a view of the interior was impossible; tension +3; vision, fingers 1 meter. Left eye: astigmatism 3 D. 90 degrees; vision, 0.3 with +0.50 D. S. = +2.50 D. C. axis 90 degrees = 0.8; anterior chamber quite shallow; no cupping of the optic nerve, but the retinal vessels showed sclerotic changes. General blood pressure was 175 systole. Elliot operation on the right eye with complete iridectomy reduced tension to subnormal. The next day the left eye, although not complained of, showed a rather widely dilated pupil. Under a miotic it quickly became contracted. During that night the patient was awakened by pain in the left eye and the head similar to that experienced in the right eye. Eserin was immediately used, so that in the morning the pupil was small; the vision still hazy, probably caused by the miotic. An Elliot operation was performed without iridectomy, as the iris did not present itself in the wound. Two days later she noticed something hanging in front of the left eye. Examination showed a subhyaloid hemorrhage covering most of the macula lutea region. Allowed to return home on the 22d, with the following result:

O. D.: vision = 0.6; c + 2.00 D. C. 90 degrees = 0.8 +; T. = 14 mm.; F. slightly contracted.

O. S.: vision = 0.1, no improvement; T. = 15 mm.; F. = normal.

Later report by letter, sees better with each eye; general health excellent.

CASE 3.—Mr. J. W. L., aged 69, seen Feb. 3, 1912, because of a blind and painful left eye, which he thought should be removed. In April, 1911, after an unexpected evening bath from the upsetting of his wagon into a pool of water, he noticed the following day the left side of his head was quite painful, or "neuralgic," as he chose to express it, and the left eye was

noticeably red. In the afternoon there was a feeling of a foreign body in the eye. In fact, he looked in vain for an eyelash. Later in the day the eyeball became extremely painful and continued so during the entire night, leaving him well used up. Consequently, he went to a hospital, remaining there ten days, and received some benefit. The vision at this time was large objects close at hand. After leaving the hospital the eye had been painful intermittently with the vision constantly decreasing.

In his past history, the following things were noted: yellow fever forty-eight years ago; pneumonia thirty years ago; and a second attack one year ago; otherwise, considered himself well, except once every month or so he had attacks of excruciating and generalized abdominal pain, with marked tenderness, always demanding the attention of a physician.

Examination: Patient plainly showed the effects of carrying a burden by his haggard and pinched expression. Vision: O. D.=0.2; O. S.=nil. Right showed no evidence of any trouble, and vision was brought up to normal with lenses. The left eye was a perfect picture of an absolute glaucoma, with its congestive, steamy cornea, shallow anterior chamber, widely dilated and immobile iris, cataractous lens, and tension 90 mm. General blood-pressure 170 systole. Operation the next day by the Elliot method with partial iridectomy reduced the tension and afforded immense relief immediately. The recovery, although rather long (twenty-five days), was ultimately good, the tension being 19 mm. There was, however, no change in vision, a thing not unexpected. Some months later, while leading a cow, the patient was struck in the operated eye by the cow's horn, causing a rupture of the sclera concentric with the limbus and 10 mm. long, the center of which passed through the trephine opening. This required an enucleation.

CASE 4.—Mrs. B. D., aged 58, was seen in February, 1912. This patient had been treated by my father three years previously for an acute attack of hemorrhagic glaucoma of the right eye, and it was my privilege to watch her at that time. Everything possible was done medicinally to relieve her. In spite of this the tension remained above normal, pain was almost constant, and vision was failing fast. In view of these facts it was deemed best to operate, and iridectomy was the choice. This was performed under a local anesthetic, and ideally executed. Just as the eye was being dressed the patient complained of a flow of hot water on her cheek. This proved to be a small stream of blood spurting up through the wound. Then quickly followed the vitreous with partial collapse of the globe, which was enucleated immediately. Nothing further was heard from her until Feb. 9, 1912, when she was brought into the Infirmary, after having suffered a week with symptoms of acute glaucoma in the other eye, the vision of which had been reduced to perception of light. Furthermore, the disease had so weakened her physical condition and changed her mental dis-

position that one could scarcely realize she was the same being. The high ocular pressure, 70 mm., was also accompanied by a high general blood-pressure, 190 systole, so that an operation was deferred to await the results of other methods, especially since the disease of the right eye had been of the hemorrhagic type. However, it soon became evident that an operation was the only hope for sight, if that was to be obtained at all. Consequently an Elliot operation was done without an iridectomy, as after the gradual release of pressure the iris did not present itself in the usual manner. On the next day the anterior chamber was two-thirds filled with blood, which disappeared in a week. Recovery was further delayed by a mild iritis. March 25, 1912, test showed vision = 0.1; with - 1.50 D. S. = 0.3 +.

June, 1914, condition noted as follows: tension 15 mm.; vision = 0.1; with lens = 0.2 —. The loss of vision was due to a central lens opacity. Her general health had been rather poor for the past six months.

CASE 5.—Mrs. M., aged 68, French. Treated in February, 1911, for an acute recurrence of a chronic glaucoma of the left eye, with the following results: vision = 0.4; tension normal. Blood-pressure 150 systole. She was unable to state exactly when the trouble began or the number of previous attacks, but was positive that vision was less after each inflammatory spell. As circumstances were such that a careful watch was impossible, an operation was advised but was rejected. The present attack had been in progress one week. The vision now was fingers at ten feet; tension 80 mm. Elliot operation Feb. 16, 1912, with a complete iridectomy, reduced the pressure to subnormal. The right eye at this time showed no evidence of trouble. Vision = 0.5 with + 2.00 D. S. = 1. Five days later, while dressing the operated eye, the right pupil was noticeably enlarged, and closer observation revealed a somewhat steamy cornea, shallow anterior chamber, and tension plus. Three days later, although miotics were being used, the tension suddenly increased to 45 mm., with slight pain and loss of vision to 0.5 so an Elliot operation with iridectomy was performed. Recovery in each case was uninterrupted, and the vision on returning home was as follows: O. D. vision = 0.3; with correction 1; O. S. vision = 0.1, not improved. Tension normal, with good ocular filtration. Seen again in June, stating that a month or so ago noticed a little redness about the right eye, which she attributed to a cold. Since then the eye has been a little red, but at no times has there been any pain; vision reduced to 0.1, due to an exudate in the pupillary area. As the tension was normal, atropin was given for home use. The last examination (June, 1914) was as follows: right eye, tension 12 mm., left eye, tension 15 mm., O. D. vision = 0.1 +; O. S. vision = fingers at ten feet. Field of vision, in O. D. normal, O. S. contracted 10 degrees. The congestion in the right eye had entirely disappeared.

CASE 6.—Mrs. A. M., aged 58. Came to the clinic at the Infirmary in September, 1912, with the complaint that the left eye had been constantly painful for three weeks. The first trouble in this eye began in 1898, and seven years later, in consequence of severe pain, she went to the Infirmary, where she was treated for three months with only partial relief, as she refused any operative measures. Upon discharge the vision was 0.2; field slightly contracted and a tension of + 2. In 1908 she again returned for treatment, at which time the vision was p.l. only, and tension + 3. The removal of several bad teeth afforded a great deal of relief for a week, and then, as there was a return of the trouble, an enucleation was proposed, but was rejected. At this time the eye was congested; cornea steamy; pupil dilated and immobile; tension 90 mm., and vision = nil. Her general health was extremely delicate, the result of an advanced tubercular lesion of the right lung. An Elliot operation without iridectomy reduced the tension to normal, and she was allowed to return to her outdoor life the next day. The right eye was not involved, and the vision was 1, with correction of compound hypermetropic astigmatism. For a while after the operation, she was free from any disturbance, and her general health improved. Soon the right eye showed glaucoma symptoms, which have thus far been controlled by non-operative measures. The left has remained quiet, no perception of light, and tension 15 mm.

CASE 7.—Mr. F. N., aged 80, has been a hat trimmer for the last fifty years, using his eyes eight to ten hours constantly each day. Several years ago the vision of the left eye was lost from glaucoma, and now it presented the characteristic appearance of an absolute glaucoma. For some time past toward the latter part of the day the vision of the right eye became foggy, so that some days, especially dull ones, it was rather difficult for him to find his way home. The next morning the sight was always clear, and it was only when this failed to occur that he was brought to the Infirmary by his wife. The vision was light perception with a tension of 75 mm. The other changes in the eye incident to an increase in pressure were present. Furthermore, an examination of his general system showed a high blood-pressure of 220 systole, well advanced arteriosclerosis, mitral regurgitation, low-grade nephritis, and a partial loss of compensation. The next day an Elliot operation with a Fox flap and partial iridectomy was performed without accident. Allowed to return home in two weeks. A test July, 1913, showed vision = 0.3 + with + 7.00 D. S. lens; good filtration with a tension 18 mm. Repeated examinations, the last of which was in July, 1914, showed exactly the same condition, and furthermore, he has been at his work, with the exception of three weeks in January, 1913, when there was complete loss of compensation. At this time the left eye became somewhat tender and painful, with tension of 95 mm. Miotics

seemed to further increase this sensitiveness, so that after the loss of compensation was restored, an Elliot operation was tried. After the trephine opening had been made, the iris bulged and ruptured before anything further was attempted. There was considerable pain and examination revealed an eyeball as hard as before the operation. The eye was watched two days, at which time it was removed to relieve his suffering.

CASE 8.—Mrs. A. T., aged 84, in September, 1910, was seen in consultation, suffering with an acute glaucoma of the right eye, of one week's duration. Some time previous to this attack her strength had become so feeble that she was unable to be about, and recently had become nervous with spells of mental derangements. In three weeks the eye quieted with a restoration of vision, until February, 1912, when there was a return of severe pain and loss of sight. Tension was 75 mm. An Elliot operation with partial iridectomy reduced the tension to minus. The next day fingers were readily counted across the room (twelve feet). The recovery was rapid and without accident. Her feeble physical and mental condition allowed only rough tests, which showed that she had good, serviceable sight with good filtration and tension 18 mm. In January, 1913, her attendant was awakened by the moans and groans of the patient, and finally learned that the left eye was painful and without sight. At times the patient acted like a raving maniac and together with occasional vomiting, the attendant reported a hard night. Furthermore, the patient refused to have anything done for her. It was with the greatest difficulty that she was quieted long enough to determine that there was an increase in intra-ocular pressure. As medicinal treatment was out of the question, she was immediately etherized, and an Elliot operation with Fox flap and partial iridectomy reduced a 65 mm. tension to a minus. She came out of the ether quietly and then passed into a sleep for fifteen hours, being awakened twice for nourishment. Her much deranged mental condition prevents any accurate tests, but she reads, and tension in each eye is normal with good filtration.

CASE 9.—Miss E. M., aged 58, consulted my father in July, 1906, because of failing sight in her right eye, the vision of which was 0.05, with the field concentrically contracted about ten degrees; tension normal. The diagnosis was an inflammation of the optic nerve and retina. The left eye was blind from a past inflammation of the uvea, and the tension was increased to +2. In September, 1909, the vision of the right eye had improved to 0.4, nearly. The left eye had been troubling her for a month and the tension had increased to +3. An iridectomy of the left eye was advised, but the patient chose to try other measures. In December, 1910, the left eye was enucleated for the relief of constant pain, as no other operation seemed justifiable at this time. In December, 1912, the vision of the right eye dropped rather suddenly to fingers at three

feet, with a tension of $+1$. Flame-shaped hemorrhages were seen scattered through the retina, but more numerous in the macula lutea region. Physical examination revealed a slight mitral regurgitation; blood-pressure 190 systole; and albumin with casts in the urine. Three months later the eye became so painful from an increased pressure, 65 mm., that an Elliot operation with partial iridectomy was performed to relieve her. A mild iritis followed, which quickly responded to treatment. Since then she has been comfortable, the tension remaining 25 mm., but the vision has not improved, although she is able to write a letter rather crudely.

CASE 10.—Miss J. S., aged 70, seen by my father in 1909, complained of seeing a rainbow around a light. Right eye, vision = 0.1, not improved because of central opacity in cornea. Left eye, vision = 0.1, with $+1.00$ D. S. = 2.50 D. C. axis 75 degrees = 0.3. The tension in each was not increased. A weak solution of pilocarpin was given for night use only. No trouble until August, 1912, when the left eye suddenly became painful, with a reduction in vision. The attack only partially improved when another came on, and this continued until her visit in October, 1912. The eye was intensely congested and painful; tension 68 mm., and vision reduced to perception of light. For two months she refused any operation, and furthermore, by spells was irrational, which interfered materially with giving her the proper treatment. Finally she consented, and an Elliot operation, with Fox flap and partial iridectomy, was performed. Excellent recovery; the vision improved to 0.05, tension 20 mm., and her mental disturbance subsided.

CASE 11.—Mrs. K., aged 60, was seen in June, 1913, with a history that two years previously the right eye had been struck by a block of wood two inches square, thrown at a distance of six feet. The lids immediately became swollen, and the eyeball was painful. In two weeks the swelling subsided and pain left. Ever since then there has been sensitiveness about the eye, and vision has gradually failed to perception of light. An examination showed a secluded pupil with the iris pushed forward; opaque lens; tension 48 mm., sensitive to touch. The right eye with correction had vision of 0.8, and a normal field and tension. In the first twenty-four hours under observation, 24 ounces of urine was passed, which showed 8 per cent. sugar, small per cent. albumin, and large number of casts. As far as could be ascertained, she had never been aware of any trouble. Later tests showed a reduction of sugar to 3 per cent. The right eye was trephined, with a partial iridectomy, and the tension was reduced to minus; recovery good. Test December, 1913: right eye, vision = perception of light; tension 18 mm.; left eye, vision = 0.4, lens cataractous. Since then numerous letters have been received, and the only complaint is that the sight of the left eye is decreasing, probably due to her general condition.

CASE 12.—H. F. D., aged 68, seen in July, 1912, because one week previously the sight of the right eye was reduced to perception of light and had remained so since then. The ophthalmoscopic examination showed evidences of sclerotic changes in the vessels with hemorrhages quite large in the macula lutea and optic disk regions. The radial vessels were hard and gave a tension of 170 systole. Urine examinations showed a low specific gravity, slight trace of albumin and a few casts. Two weeks treatment brought about an improvement in every way, so he was allowed to return home under the care of his family physician. In January, 1913, he returned with the right eye painful; tension 98 mm.; pupil widely dilated; no red reflex, and no perception of light. As the eye was sensitive to light, an enucleation was advised, but he wanted to save the eyeball if possible. So a trephine was tried, but no sooner had the opening been made when a small column of blood spurted up through it with a little watery vitreous, and then the eye became hard and painful, so much so that it was removed two hours later.

CASE 13.—Mr. V. G., aged 72, an army veteran, with poor health, was seen in April, 1913, because the vision of the right eye within the last two years had been failing, so that now he saw fingers at four feet. His left eye had been blind several years. No other symptom called his attention to his eyes. The tension of the right eye was 50 mm., left eye 60 mm. Field of vision of right eye contracted about 15 degrees. There was a cupping of the disk of 3 diopters in the right eye and 5 diopters in the left. In fact, the eyes were well advanced cases of simple glaucoma. An Elliot operation, with Fox flap and partial iridectomy, was performed on each. Mild iritis followed both operations. Six weeks after, right eye vision=fingers at fifteen feet, tension 15 mm.; left eye vision=perception of light, tension 18 mm.; field of vision in the right eye nearly normal. One letter received in June, 1914, reported improvement in vision of right eye, and also better general health.

CASE 14.—P. T., aged 48, in April, 1913, consulted me to ascertain if anything could be done to help the vision of the right eye. The first trouble in this eye was felt four or five years previously, when there was pain and dimness of vision. This condition disappeared, and since then he has had many such spells. The vision was perception of light only; tension 55 mm.; scleral vessels markedly enlarged; pupil widely dilated, and a cupping of the optic disk of 5 diopters. The left eye had been blind some years. The examination showed dilated scleral veins; no anterior chamber; pupil small, secluded and occluded, and tension about normal. Upon lifting the upper lid, a large equatorial staphyloma came into view. Blood-pressure 115 systole. Urine examination and Wassermann test negative. An Elliot operation in the right eye, with Fox flap and partial iridectomy, reduced the tension to 20 mm., but did not improve vision. The left eye was enucleated, and was about one and one-half times

as large as a normal eye. In its equatorial region a ring-like bulging extended around the ball. Future tests of the right eye showed the condition as above noted.

CASE 15.—Mr. S. A., aged 70, was seen in July, 1911, with the history that four years previously his vision suddenly became dim, lasting until the following day. Several such spells had occurred up to the present time, with a gradual loss of vision after each. No pain felt until two weeks ago, and since then it has been rather constant. Both eyes congested and sensitive to light. Vision, right eye, fingers at two feet, left eye, no perception of light; tension, right eye, 45 mm., left eye, 90 mm. Field of vision contracted 20 degrees. Blood-pressure 180 systole. Treatment improved the vision of the right to 0.1, and lowered tension to 25 mm., while no change was made in the left eye. A view of the fundus of the right eye showed a cupping of 3 diopters. An Elliot operation, with Fox flap and partial iridectomy, did not lower the tension of the left eye, on account of an intra-ocular hemorrhage. Six days later, on account of the continued pain, in consequence of which there was a great loss of strength, the eye was enucleated. The eye contained a blood clot with some fluid vitreous. The choroid showed evidences of sclerosis. This enucleation produced a favorable effect on the right eye, so that under non-operative treatment the tension is normal, and vision is 0.1 +.

CASE 16.—Miss X. L., French, aged 68, noticed for some months dim morning vision, which cleared after being about an hour or so. Now, May, 1913, the vision is failing fast. Examination: right eye, vision = 0.3; with -1.00 D. S. = -0.50 D. C. axis 180 degrees = 0.3 +; left eye, vision = 0.1; with -1.00 D. S. = -1.00 D. C. axis 180 degrees = 0.2. Add + 3.50 for reading. Field of vision narrowed in right eye 10 degrees, left 15 degrees. Tension, right eye, 2 +; left eye, 2 +; somewhat steamy and insensitive corneas; anterior chambers shallow; pupils moderately dilated and sluggish to light, and cupping of the optic disk of 3 diopters. An operation was strongly advised, because the patient did not seem to grasp the severity of the situation, and probably would not carry out the treatment systematically. However, she deferred having anything done at this time. In August, 1913, I was called to her home and found her suffering with a painful left eye, which had been so for a week. The vision = nil; tension 85 mm. The patient was extremely weak and nervous from her long-continued suffering. An Elliot operation, with Fox flap and partial iridectomy, reduced tension to minus. As she lived out of town, the after care was left to her family physician. Four weeks later, the right eye became painful, with a tension of 78 mm. and a reduction of vision to perception of light. An Elliot operation, with Fox flap and partial iridectomy, reduced tension of this eye to minus. The left eye showed a good filtration, tension 17 mm., vision = fingers at six feet. Later report from her family

physician as follows: right eye, vision = fingers fourteen feet, tension normal; left eye, no change.

CASE 17.—Mrs. F. K., aged 51, French, came to the Infirmary in October, 1913, suffering with a painful right eye. The cornea presented an unusual appearance, in that apart from the general haziness there was a denser medial horizontal band 3 mm. in width. The vision was nil; tension 80 mm., with marked redness, part of which, no doubt, was due to an inflammation of the lacrimal sac, from which a large amount of pus could be expressed. Treatment was immediately instituted for both conditions. In a week, miotics, although quite painful, improved the ocular condition, so that the cornea became less hazy, and the tension was reduced to 45 mm. Just at this time sickness in her family made it necessary for her to return home. Later, in December, 1913, after passing through the strain of a death in the family, she returned, with more pain and a tension of 80 mm. At this time the dacryocystitis had subsided, so that smears were negative. Under ether an Elliot operation, with Fox flap and complete iridectomy, was followed by an intra-ocular hemorrhage, so that the tension was not materially changed. This absorbed so that ultimately the tension became 30 mm., with good ocular filtration, but the vision remained nil.

CASE 18.—H. H. H., aged 47, had slowly failing vision for ten years previous to December, 1913, so that he had to give up his position as a lumber surveyor. He never had any pain or redness of his eyes. The vision of the right eye was fingers at ten feet, that of the left, fingers at two feet. The field of vision concentrically contracted in the right about 10 degrees, and in the left 20 degrees; cupping of the disk in right eye 3 D., left eye, 5 D.; tension of right eye 35 mm., left eye 45 mm. The right eye was operated upon first by the Elliott method, with Fox flap and partial iridectomy. This improved vision to 0.2 + and reduced tension to 12 mm. Later the left eye was operated on by the same method. Everything went along ideally for seven days, when he developed an acute conjunctivitis in each eye. The left became infected, and in spite of prompt and energetic treatment, it was lost for visual purposes. A recent letter from him states that he is able to do his work better than for some years.

CASE 19.—J. B. J., aged 53, treated for two months in the fall of 1913 for a recurrent attack of iridocyclitis of the right eye. The margin of the pupil was completely adhered to the anterior capsule of the lens and there was a tendency toward an increase of intra-ocular pressure. Vision equaled fingers at three feet. Positive history of syphilis contracted several years previously, although the Wassermann test was negative. In December, 1913, returned, with more pain in the right eye, which was very sensitive to light and pressure. Tension 70 mm. Anterior chamber two-thirds filled with blood in layers of different colors. Blood-pressure 170 systole. Three weeks'

treatment showed no appreciable change except fresh hemorrhages in the anterior chamber. As the eye was blind, he wanted to have it removed, but an Elliot operation was substituted. In excising the iris, the patient squeezed and caused a slight loss of watery straw-colored vitreous. Recovery was good, but rather slow. In June, 1914, the tension was 45 mm., uveal tissue in the opening, no perception of light and occasionally painful.

CASE 20.—Mrs. B., aged 70, was long a sufferer of articular rheumatism, being unable to walk without the aid of crutches. Twenty years previously had the right eye iridectomized for the relief of acute glaucoma. Now the tension is normal, and vision equals 0.5 with the correction of 4 diopters of hypermetropia. Ten years ago the other eye had a similar attack, but her physicians refused to operate on it. Again five years ago she had another severe time with the eye, which finally subsided under miotics. For the past winter the vision of the eye became foggy by spells, requiring the use of a 1 per cent. solution of eserine to clear away the mist. Finally, in the train, on her way to consult me, the eye suddenly became painful, accompanied with nausea. When examined, the eye was in the midst of an acute attack of glaucoma, with vision reduced to perception of light only, and tension 85 mm. Blood-pressure 185. Well marked mitral regurgitation. The eye responded poorly to treatment, so in the interest of vision an Elliot operation, with Fox flap and partial iridectomy, was performed. Two weeks later, at the time of her discharge, vision = 0.5; tension 15 mm. Letter in July she reports her eye comfortable with some improvement in vision.

CASE 21.—Mrs. M. G. H., aged 57, first seen in September, 1913, with a cataract in the left eye; the right lens clear, vision 0.6, and with correction, 1 mostly. Her general health was poor; blood-pressure 190 systole. Under a month's treatment there was an improvement in general health with a reduction of blood-pressure to 160 systole, so that a simple extraction was performed without accident. Recovery was prolonged by an attack of iridocyclitis, lasting three weeks. During this time no increase in intra-ocular pressure was noted. Vision of this eye on leaving was 0.6 with correction. Three weeks later, while about her housework, the eye suddenly became painful, and increased in severity to such an extent that she went to bed, remaining there the greater part of six weeks. Living some distance away, she was treated by her family physician, who first used atropin, but soon stopped, as the eye became worse. Finally she felt able to come to see me, at which time her eye was but slightly congested, cornea very hazy, anterior chamber quite deep, pupil 4 mm. in diameter, tension 98 mm., and blood-pressure 200 systole. Treatment reduced the tension to 68 mm., and finally to 40 mm., vision remaining nil. The cornea cleared, and an examination of the fundus showed a marked cupping of the optic nerve. Later an Elliot operation, with Fox flap and with-

out iridectomy, further reduced the tension to 20 mm., but there was no change in vision. Last examination, in June, 1914, showed the eye in exactly the same condition as when examined after the operation. Furthermore, the patient had been able to attend to her household duties without any disturbance.

DISCUSSION

DR. WALTER R. PARKER, Detroit: I wish to congratulate Dr. Holt on the care with which he has worked up his case reports; also on the good results obtained. I tabulated one hundred cases out of one hundred and thirty-five operated on by Col. Elliot, also twenty-seven of my own cases, and Dr. Holt's results are the best both as regards good results obtained, and in the number of cases showing signs of iritis.

I believe that the sclerotrephining operation which Col. Elliot has given us offers a method of procedure that is to take a permanent place in the treatment of glaucoma. I am convinced, however, that no one procedure is applicable to all cases and that the particular type of the disease best treated by trephining has not yet been definitely determined.

It would also appear from the conflicting reports that the exact mode of procedure has not been fully determined. Most of the systematic reports show, for instance, that iritis follows less frequently when a complete iridectomy is performed at the time of the operation. It may be as Col. Elliot suggests, the iridectomy has nothing to do as a factor in the reduction of the tension, but it appears to have a decided influence on the amount of reaction following the operation. The results obtained from the classical iridectomy in cases of acute or subacute glaucoma should make one hesitate before adopting other methods unless there is some contra-indication for its use. In simple cases the trephining operation finds its best field. Even here it may prove to be better to use myotics as long as the excessive tension can be held in check, or to do an iridectomy in cases where the anterior chamber is not too shallow, and reserve the trephining operation for those cases in which other methods of treatment have failed, being careful not to wait too long. The tonometer affords such a good control of these cases that intelligent waiting is now made possible.

One of my patients had simple glaucoma with acute exacerbations. Vision in each eye was 6/15. The patient had been under miotics for several months, but the disease was progressing. I did a corneoscleral trephining operation without accident. The chamber remained empty for nineteen days. When it did reform the iris showed signs of inflammation and in spite of the fact that atropin had been used regularly, adhesions formed and at the end of four weeks' time the pupil was occluded and the vision was reduced to light perception. I later did a deep iridectomy on the other eye and the vision has held, the tension is normal and all symptoms have disappeared. But this is only one case.

I do not wish to be understood to be opposed to the corneoscleral trephining operation in certain cases, but until we know more about the etiology of glaucoma I doubt if it is advisable to use any one procedure in all cases.

DR. H. GRADLE, Chicago: I was very glad indeed to hear Dr. Parker's remarks, because at the Illinois state meeting in the spring and the Wisconsin state meeting this fall, I emphasized the fact that I believe in acute and subacute glaucoma the classical iridectomy of von Graefe was the only method I felt justified in employing. As long as the results of the trephining exist, that patient is subject to the dangers of a late infec-

tion. We know that when there exists an infection of the conjunctiva by the pneumococcus or streptococcus these germs penetrate into the subconjunctival tissue and thence to the fistula, where they can enter the eye. Consequently, I believe we should hold trephining as an operation of last resort. Another feature of trephining I have observed in three cases, one with and two without the so-called quiet iritis, is an obliteration of the field following the iridectomy. Trephining results are good, but it is an operation of danger we should keep as a last resort.

Dr. Jobson, Franklin, Pa.: We learn from our failures as well as our successes. One of my failures was a case of late infection, following ten days after what was up to that time a successful trephining operation for glaucoma. The patient was nauseated on the tenth day after the operation and vomited frequently for about twelve hours. Temperature 103 F. Severe pain in the operated eye, which was greatly swollen and reddened. Iridocyclitis followed the next day and lasted for about four weeks, but was finally controlled by vigorous treatment. But vision was greatly impaired. A small ununited piece of flap was discovered with a magnifying glass, which was probably the point of entrance for the infection. The treatment was commenced by injecting 1:5,000 bichlorid of mercury solution under the flap at this point. Mercury and salicylates were given freely, and a solution of atropin and dionin was used locally.

In another case the eye had to be enucleated, as the sudden relief of pressure caused severe hemorrhage from the depths of the eyeball, and this destroyed it.

Dr. Wm. O. Bonser, Toledo: There has been considerable hesitancy displayed relative to the acceptance of these bodies as being of diagnostic value in the identification of suspected cases of trachoma. One of the principal reasons for this was due to the fact that observers reported the presence of cell inclusions in various other diseases of the conjunctiva. It was due to this diversity of results that Lindner brought out his classical work on this subject in 1912. He proved two out of three of the axioms of Koch's laws, viz., these trachoma organisms when inoculated into the conjunctiva produce trachoma. These organisms are present in cases of trachoma, especially in the acute stages.

The third law of Koch, according to Noguchi and Cohen, *Archives of Ophthalmology*, March, 1914, relative to these bodies, has been filled, for they have succeeded in growing these organisms outside the body.

The methods for staining these bodies which we employ is not a complicated one. The conjunctiva, after cocainizing, is gently scraped with a blunt eye spud and an even smear made of some of this material, which contains some of the epithelial cells of the conjunctiva. The smears are then dried in air and fixed with equal parts of absolute alcohol and ether for thirty minutes, and then are stained with Giemsa's stain, which must be freshly prepared. We have found it more satisfactory to use a weak solution of the stain and stain over a longer period.

One must have familiarity with the diversity of forms that these trachoma bodies can take and one must be able to distinguish this cell inclusion from degeneration occurring physiologically in the epithelial cells.

However, for diagnostic purposes, this bluish area, as shown in Chart 1, of compact granules near the nucleus, is characteristic and the technic can be done with a very simple laboratory equipment.

We have found the presence of these bodies of diagnostic value in distinguishing trachoma from cases of pseudotrachoma, where the diagnosis could not be made clinically.

SOME OBSERVATIONS OF THE EYE CLINICS OF PARIS

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At the Lairboisiere is Morax, who operates beautifully, and as we would expect from one who has written so extensively on the bacteriology of the eye, with the most careful attention to asepsis. I saw him do several cataract extractions, entropion and other lid operations, lacrimal-sac extirpations, and in one case of dacryocystitis he enlarged the opening into the nose by chiseling through the nasal process of the superior maxilla anterior to the lacrimal ridge, and into the bony canal thus formed he pushed the lower portion of the sac, which had previously been dissected loose but left in situ. In common with all the operators whom I saw in Paris, he destroys the mucous membrane of the nasal duct with the actual cautery after the lacrimal sac is removed.

A very useful adjunct for teaching purposes in the operating room was a frame about 3 by 3½ feet, containing a plate of translucent glass, behind which were colored outline representations of various parts of the eye. This allows a sketch to be made quickly when needed in explaining an operation.

The eye instruments, especially the cutting ones, are sterilized by dry heat. This method is the one adopted at all the eye clinics I visited in Paris. The instruments for each operation are placed in a metal box especially designed for this assortment of instruments. The box is then placed in the sterilizer, which is usually heated by electricity and provided with a thermometer and often a thermostat. When a temperature of 130 degrees centigrade is attained, the current is turned off, but the heat usually increases until 140 degrees is reached before it begins to decrease. The sterilizer is not opened until cool, the process requiring about two hours. The instrument boxes are opened when the surgeon is ready to operate. They maintain that knives and other cutting instruments remain sharp much longer than when boiled. They certainly retain their polish much longer.

The needles used in eye surgery are placed in boxes after being threaded, the several layers being separated by layers of paper. The dressings for each patient are placed in a separate metal box.

A most interesting room in the refraction department of this clinic is the one where they examine the cases of paralysis of the external ocular muscles. It is 5 meters square and quite dark. On one wall are painted nine white squares and at the meeting point of the lines which form these squares are candle-shaped electric lamps with frosted glass, controlled from the other end of the room, where the surgeon and patients stand, about 4 meters from the wall with the squares. The lamps may be switched on successively by the examiner. One of the patient's eyes is covered with a colored glass disk and he is then asked to describe the respective position of the white and colored lights which he sees. His answers are recorded graphically on gummed slips of paper for ultimate insertion in the notes of the case.

The out-patient department which consists of a large, well-lighted waiting-room with iron furniture painted white, is divided into two parts by a partition, one side of which is for the patients suffering with external eye diseases and the other those with intra-ocular diseases, and patients for refraction.

In the department for external diseases are two small chambers, where patients with infective diseases may be kept apart, especially those with whooping-cough or measles. The presumably septic cases are dealt with in a special consulting room to which is attached a small laboratory for the rapid examination of specimens. More complete examinations, calling for cultures, inoculations, etc., are conducted in the research laboratory, which is easy of access from the small laboratory. The non-septic cases are dealt with in another room, from whence patients, if necessary, are passed on to the refraction department.

Magitot is associated with Morax at this hospital, but I think he devotes most of his time to teaching and laboratory work. He photographs the external eye diseases in colors on glass plates which are used by trans-illumination for demonstration to the students, thus avoiding, he stated, the discomfort to the patient of being handled by a number of men, some of whom may not be as gentle as they should be. This photographic work is done in the eye department of the hospital. The

laboratory is one of the best in Europe and the work done is most thorough in every way.

The Landolts, father and son, have their private clinic. The son was away at the time of my visit, but the father, Dr. E. Landolt, was most gracious. He demonstrated for me with the greatest care two advancement operations. You know that he operates on both eyes at the one sitting, as has always been his custom, and does not believe that tenotomy has a place in ophthalmology. He advances the muscle to the corneal margin resecting all or part of the tendinous portion and often some of the muscle. He is very careful to see that the muscle is separated from its sheath and that its fibers are exposed, also that the sclera is bare, all episcleral tissue being removed, thus facilitating union. He keeps both eyes bandaged for eight days, removing the stitches on the sixth day and the dressings daily for cleansing purposes.

In both of the operations I witnessed there was a marked overcorrection; this, he said, was customary and would right itself. When the dressings are removed, he has the patients wear correcting glasses, which are tinted to prevent the dazzling due to the mydriatic, which is continued for some time. He seemed to have no fear of enophthalmos, which Bishop Harman insists occurs when an internal or external rectus is shortened without relaxing its opposing fellow. You will remember that Mr. Harman accomplishes this in his reefing operation by making his jig saw incisions in the muscle opposing the one he reefs or tucks.

Landolt demonstrated his fixation forceps and his eye speculum which lies on the side of the nose and is thus out of the way when operating on an external rectus or making an incision for cataract. He also showed me a capsule forceps for secondary cataract operation, one blade of which is plunged through the capsule with the forceps open and then closing them, thus catching a portion of capsule and removing it with the forceps.

His remarks were lucid and often quite pointed, as, for example, when he said, "At the close of my career I make all of my cataract operations with a large incision at the limbus, make my cystotomy, then, while the view is not obstructed by blood, and then make a large iridectomy." Again to my query, "What do you think of the intracapsular, or Smith operation?" he replied, "I saw several eyes emptied by that method in Paris a few years ago."

I suggested that I had tried the preliminary capsulotomy, as recommended by Homer Smith, with very good success. He said that he was unable to see the advantage of it.

The Quinze Vingts, which was founded A. D. 1254 to 1460 for fifteen score of blind men, has an unlimited amount of material which is handled in the out-patient department with almost startling dispatch, from thirty to fifty patients being examined by one physician in an hour, the doctor using focal illumination in most of the cases. The diagnosis of external ailments are recorded on the patient's card with notes by a clerical assistant at the time. The straightforward cases are prescribed for at once, the prescriptions being printed on slips. Those requiring ophthalmoscopic examination or refraction are referred to these respective departments.

It was my pleasure to witness Chaillous, Valude and Kalt operate on different days, the operations comprising those usually seen in a large clinic. It was most interesting to observe the different methods of these three operators, probably men of equal ability. This was particularly noticeable in cataract extraction. Chaillous makes a rather large incision at the sclerocorneal margin, leaving a bridge of conjunctiva above and slightly to the nasal side, and usually making an iridectomy. Valude makes the same incision leaving a large conjunctival flap; he made an iridectomy when I saw him, although I understand he usually does not do so. Kalt makes his corneal suture with a specially prepared cotton thread, forcing the needle into the cornea horizontally near the limbus, then passing it through the conjunctiva and episcleral tissue very near to the cornea, leaving a loop between the two, through which the Graefe knife passes in making the incision. He then makes a capsulotomy with his well-known capsule forceps, and after the lens substance is extracted and the iris replaced, he tightens the loop and ties the two free ends. He does a simple extraction. I was much interested in this, having had difficulty in removing this stitch. Dr. Kalt cuts it with a special scissors and has no trouble in removing it. These surgeons, in common with all whom I saw making cataract operations, remove any lens or capsule fragments remaining after the expulsion of the lens with Teale's suction apparatus.

The ophthalmologists of Paris stitch the conjunctiva over the cornea much more frequently than we do. In one month I witnessed this eight times in perforating wounds and ulcers.

In glaucoma the Elliot trephining operation seems to be the one of choice, but was always combined with an iridectomy. Only twice did I see an iridectomy made for this ailment without trephining.

At the Rothchild's Foundation, which is one of the largest and best-equipped eye hospitals in the world, I saw many operators, some exceedingly good. It was here that I saw Millée making cataract extractions with no instrument save the Grafe knife. He held the lids apart with his fingers, making his cystotomy as the knife passed through the anterior chamber, finishing the incision with a large conjunctival flap. He expelled the lens substance with his fingers and the lid margins. In only one of the three extractions I witnessed did he use a spatula to replace the iris. He finishes the operation by a stitch passed through the conjunctival flap and the adjacent conjunctiva and episcleral tissue.

I visited Professor Gauché at the St. Louis Hospital to hear his views on the treatment of eye diseases of luetic origin. He very decidedly denounced the use of salvarsan and neosalvarsan as both useless and dangerous, and uses a solution of benzoate of mercury by deep muscular injection. The same day at the Quinze Vingt I witnessed the intravenous injection of neosalvarsan by the use of a hypodermic syringe, the usual dose being dissolved in only 2 c.c. of sterile distilled water, the patient leaving the clinic a few minutes afterward. Truly many men of many minds.

Darier, who, like Landolt, conducts a private clinic, is always interesting and one cannot visit him without carrying away much of his therapeutic optimism. He treats over three thousand new patients yearly, operating on them as they present themselves; even his cataract patients are permitted to go to their homes immediately after the operation.

He was trying a new treatment of high myopia one day when I visited him, using small rubber cushions filled with air and having adjustable valves. These were applied over the closed lids with a few layers of gauze and a bandage applied quite tightly, the cocks being placed so the patient could open the valve and release some of the pressure if it became too painful. In addition to this, he uses subconjunctival injections, given with a curved needle posterior to the equator of the eyeball. He does this by having the patient look downward and to the opposite side, then by raising the upper lid the needle is introduced

subconjunctivally as far from the limbus as possible. He uses no instrument but the syringe. To American ophthalmologists Darier is probably most often associated with intravenous injections. The writer has been much indebted to him for his suggestion of the use of cyanid of mercury by this method. When I saw him he was using enesol, which is mercury salicylarsenate, using 1 or 2 c.c. of a 3 per cent. solution.

Dr. Darier is very insistent on the use of tuberculin in eye diseases when indicated. While he seems to prefer the Russian preparation known as endotin, if this is not well borne or does not prove efficient, he uses one of the other varieties. He uses his protargalogue and zincage with much success. I had regarded these as largely suggestive measures, but since my return have tried them with gratifying results.

At the Hotel Dieu I saw Professor de Lapersonne operate and watched his examination and demonstration of cases. It is to him the sterilization of instruments by hot air as well as the design of many of the numerous metal containers for eye instruments is accredited. The hospital impresses you as having been especially arranged with the object of making everything inconvenient, and I could not help thinking how much de Lapersonne had accomplished under so unfavorable an environment.

The nursing never, and the assistants seldom, meet the requirements of our American clinics.

In every place I presented my card I was received with the greatest courtesy and in several instances the details of methods in which I was interested were explained to me through an interpreter.

ACQUIRED NON-TRAUMATIC CATARACT OF THE YOUNG

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The question of intranasal deformity as a causative factor in the production of ocular lesions has been recognized for a number of years, and much has been written on this subject as it applies to both ocular and oral disturbances. Little has been written, however, as to the relationship between acquired non-traumatic cataract of the young and intranasal pressure.

During the last five years eleven of such cases in various stages of development, from slight opacity of the lens or capsule to complete cataractous condition, have come under my observation. Such measures were instituted as in my judgment seemed to offer the best results in each case.

A brief description of these cases, together with the line of treatment instituted, the results attained and conclusions as to the underlying etiological factor in their production, is the object of this paper.

Before beginning a description of these cases, permit me to review briefly the nerve relationship of the nasal cavity and ocular structures.

It is a well-known fact that the trifacial anastomoses with more of the cranial nerves than any other. Its relation with the sympathetic ganglion is very close. I shall only speak of the association of these parts by way of the vasomotor or sympathetic nerves in their influence on nutrition, resulting in the conditions found in these several cases.

The gasserian ganglion, as it lies on the upper surface of the petrous portion of the temporal bone, just at the exit of the internal carotid artery from that structure, divides into three branches, the ophthalmic, superior and inferior maxillary nerves, the first two being sensory, the latter motor. The ophthalmic and superior maxillary branches, which are of interest in connection with this subject, supply the ocular and nasal structures respectively with sensory impulses. The sympathetic system anastomoses abundantly with these sensory nerves through their ganglionic centers, the ciliary or ophthalmic; sphenopalatine or

Meckel's, otic and submaxillary; the first two only being of importance to us in this connection.

The superior cervical sympathetic ganglion, which connects with all the spinal sympathetic nerves, gives off a superior branch, which travels along with the internal carotid artery, passes into the carotid canal, where it divides into two branches, one passing along the external and the other the internal walls of the artery. The external, in addition to supplying filaments to the artery, forms the carotid plexus; the internal likewise gives off filaments to the artery and passes on, forming the cavernous plexus; the carotid plexus here communicates with the gasserian and sphenopalatine ganglions. The cavernous plexus anastomoses with the third, fourth, fifth and sixth nerves and the ciliary ganglion. Both the carotid and cavernous plexuses supply filaments which traverse the carotid and its branches to their final termination.

The ciliary ganglion, situated external to the optic nerve in the orbit, receives branches from the nasociliary and carotid plexus and sends branches to the interior of the eye, and through the nasal nerve reaches the upper and anterior portion of the nasal cavity.

The sphenopalatine ganglion, situated in the sphenomaxillary fossa, receives branches from the superior maxillary, the facial and the carotid plexus and sends fibers to the turbinated bones and palate. The otic and submaxillary ganglions, while connected with the third branch of the fifth nerve and the carotid sympathetic, are not of interest in this connection.

The close relationship of the sensory and sympathetic nerves, and the fact of the sympathetic nerves being also vasomotor in function, gives to them a controlling influence over nutrition.

In taking up these several cases much of their history has been omitted for the sake of brevity, only the essentials in each case being given.

CASE 1.—Boy, 18 years of age, came to me with a history of having first noticed a blurring of vision of right eye about one year previous. Six months later a similar condition was noticed in the left eye.

On examination I found vision in right eye 20/40, left eye 20/30, with several punctate opacities on the posterior capsule of both lenses, more pronounced on the right. Subjective symptoms negative, excepting blurred vision.

Personal history good, family history fair. Fundus examination showed normal condition. On making nasal examination a pronounced 'septal thickening was found making pressure

on both middle turbinates. Operation was advised, which was rejected by parents. He was put on KI treatment for four months, at which time examination showed the opacities markedly heavier. An operation was then agreed to.

A submucous resection of the thickened portion of the septum was done. The KI treatment was continued with marked improvement in three months. Three months later the opacities had entirely disappeared and vision was 20/20 in both eyes. Two years later there was no evidence of a return of the condition.

CASE 2.—Boy, 17 years of age, referred to me by a physician from a rural district, with a history of beginning failure in vision of both eyes five years previous. When seen by me could count fingers at six feet.

Family history negative; personal history, no severe acute sickness or ocular disturbance preceding the beginning failure of vision. Both pupils presented a light gray appearance, showing advanced cataractous lenses. No kidney, specific or rheumatic history.

On examining the nasal cavity a septal thickening was found pressing on both middle turbinates. A septal operation was done; also the posterior third of both middle turbinates was removed, disclosing a purulent anterior ethmoiditis on both sides.

The patient was then put on mercury and potassium iodid treatment for six months with no appreciable change. I then did a lens extraction of the left eye, which gave 20/30 vision with plus 10.00 spherical correction. Nothing has yet been done for the right eye.

CASE 3.—Young woman, 23 years of age, family history, general rheumatic condition of several members of family, otherwise negative. Personal history, eighteen months previous to coming to me she had noticed a blurring of vision in both eyes, which had gradually increased until her vision at that time was 20/40 both eyes. Both lenses showed a light-gray color, more intense in the center.

On examining the nose the posterior end of both middle turbinates was found to be quite large, making pressure on the septum. These hypertrophied ends were removed, which gave free drainage and ventilation to both nostrils. She was then put on KI and continued for six months. Vision at the end of that time was 20/25 both eyes. The case was afterwards lost sight of.

CASE 4.—Young man, 25 years of age, came to me for gradual failure of vision of both eyes, which had first been noticed two years previous. At time of first seeing him he could count fingers at ten feet; the lenses were decidedly opaque, both eyes much alike. Personal history, acute sickness of childhood, no severe sickness since that time. Had always lived in the country.

On making a nasal examination a septal spur was found on both sides, extending and pressing against the anterior two-thirds of the middle turbinates. A submucous resection of the septum was done, and after healing he was given constitutional treatment for a period of eight months, at which time his vision in both eyes was 20/60, and despite continued treatment has remained about the same.

CASE 5.—Woman, 31 years of age, living in the country; was married and had three children, the youngest of which was 6 years old. Had first noticed a blurring of vision in left eye three and one-half years before, followed six months later by similar condition in right eye. At time of first visit had light perception of left eye and could only count fingers at five or six feet with right eye.

Nasal examination showed posterior third of both middle turbinates very much enlarged and pressing on septum, with mucopurulent secretion in posterior nares. The hypertrophied portion of the middle turbinates was removed. She was then given constitutional treatment for eight months with apparently no benefit. A lens extraction was then done on both eyes, with three weeks intervening. With plus 9.00 spherical correction she is able to see, right eye 20/30, left eye 20/40.

CASE 6.—Boy, 19 years of age, poorly nourished, came to me with a history of having noticed a beginning failure of vision in right eye six years previous. Same condition began in left eye one year later. Examination showed only light perception in both eyes.

This patient gave a history of having been struck on the nose while playing ball about one year before trouble began in right eye. On examining the nose I found the septum very much thickened and deflected, making pressure on both middle turbinates, with polypi showing under anterior end of turbinates.

A septal operation was done and the polypi removed. He was then put on Hg and KI for several months with no appreciable results. Lens extraction was then done on the left eye, and with a plus 8.00 sphere gave 20/40 vision. Operation on right eye was refused.

CASE 7.—Boy, 12 years of age, was referred to me with a history of failing vision of right eye for one and one-half years, left eye two years. Family history good; personal history, measles and whooping-cough before 8 years of age.

Examination showed shallow anterior chamber, lens slightly but uniformly hazy, vitreous cloudy. Vision, right eye 20/30, left eye 20/50. Further examination showed enlarged faucial tonsils, adenoid vegetation in posterior nares, anterior third left middle turbinate greatly enlarged, crowding the septum over against the right middle turbinate. Adenoid and tonsils were removed; later a part of left middle turbinate was removed, relieving the pressure in both nostrils. Eight months later under

constitutional treatment vision in right eye was 20/20, left eye 20/30.

CASE 8.—Boy, 14 years of age, was referred to me for information regarding a "blind school." At that time lenses of both eyes were cataractous; could count fingers only at three feet.

Family history uncertain; personal history, no severe sickness in childhood, had a blow on the nose when 8 years of age. Dimness of vision was first noticed five years ago, gradually progressing until the condition stated above on first examination.

Examination showed a greatly thickened nasal septum, with practically no breathing space on either side; posterior nares filled with adenoid growth. Growth was removed from posterior part of the nose and a septal operation done. Constitutional treatment was instituted for several months with no appreciable results. The lens of right eye was then removed and with correcting lens gave 20/40 vision, some capsular involvement still remaining. Nothing has yet been done for the left eye.

CASE 9.—Girl, 18 years of age, came to me for failure of vision in left eye, first noticed four months previous. Family history fair; personal history, acute sickness of childhood, no severe illness for the last five years. Vision of left eye 20/40, right eye 20/20.

Nasal examination showed septal deflection to left side with enlarged left middle turbinate. Operation for correction of this deformity was refused. She was then put on KI and continued for four months, with result of continued failure in vision of left eye. Operation was then agreed to. The septal deformity was corrected, constitutional treatment continued and in two months some improvement was noticed. In six months vision had improved to 20/20, at which time patient moved away.

CASE 10.—Man, 35 years of age, came to me complaining of inability to see clearly for the last two months; there seemed to be a mist before the eyes.

Family and personal history good. Vision in both eyes was 20/30. Examination of both lenses showed a hazy condition. There had been no acute sickness or specific trouble.

On examining the nose the posterior ends of both middle turbinates were found enlarged, pressing on the septum. These enlarged parts were removed, the patient put on constitutional treatment and in four months vision in both eyes was 20/20.

CASE 11.—Man, 27 years of age, referred to me with pronounced opacity of both lenses. Trouble began four years previously with slight haziness and had grown progressively worse until at time of first visit could count fingers at five or six feet. About one year before this trouble was first noticed he had been struck on the bridge of the nose while playing football, which gave him considerable trouble at the time, but apparently he had entirely recovered.

On examining the nose a very much thickened and twisted septum was found, making pressure on both lateral walls. A

submucous operation was done, removing the greater portion of cartilaginous and bony septum. This was followed by constitutional treatment for six months with no improvement. Lens extraction was then done on both eyes, with an interval of six weeks; both capsules were involved in the cataractous condition, so that with correcting lens, right eye 20/50, left eye 20/60; vision was the best that could be developed.

In all of these eleven cases syphilis, nephritis and tuberculosis were not manifest; neither was there any serious acute sickness preceding these ocular disturbances.

SUMMARY

Six of these cases, ranging in age from 12 to 35 years, showed marked improvement following nasal operation and constitutional treatment; four had 20/20 vision in both eyes, one 20/25 vision in both eyes, the other 20/60 vision in both eyes, the latter poor vision being due to marked capsular involvement.

Five of these cases, ranging in age from 14 to 31 years, had disturbance of vision ranging from three to six years' duration. In none of these did nasal operation and internal treatment produce any appreciable results. Lens extraction, however, gave fairly useful vision.

Thus the conclusions to be drawn from conditions of this nature produced by intranasal pressure are, that if corrected by suitable operative procedure within the nasal chamber, before a marked destructive process has taken place in the lens and capsule, good results will follow. It is likewise evident from these other cases, which were not benefited by operative procedure in the nasal cavity, that a permanent structural change had taken place and that nasal operation was not followed by any beneficial results.

I do not wish to be understood from the above statement to specify an arbitrary time when such conditions will or will not improve following a correction of an existing nasal trouble, for different conditions may alter the time when such changes take place; but that where such a condition has existed a sufficient length of time to produce a structural change, resolution will hardly take place even after normal nutrition has been reestablished.

In view of the fact that all nerves possess trophic fibers, it is evident that where a degenerative process of the nerves themselves takes place, the trophic nerves will also degenerate and lose their function. Thus a continued pressure within the nasal

cavity of long standing will be followed by an atrophic condition of the trophic nerves, which so interferes with the nutrition of the crystalline lens and capsule as to produce a permanent derangement.

This same condition of pressure for a short period of time, however, is not likely to produce any real tissue change and when corrected will soon reestablish normal function and nutrition; while long continued pressure or irritation will in time produce actual destruction. In the beginning, pressure symptoms within the nose will produce an irritation of the sympathetic nerves which will be followed by temporary exhalation of the vasomotor nerves, while if this condition is long continued, these trophic nerves will atrophy and thus destroy the control of nutrition to the parts involved.

It seems obvious that disturbance of nutrition in the crystalline lens and capsule may take place simultaneously from the same cause, and that the resolution of one is usually accompanied by a resolution of the other.

Loss of cellular elements of the lens capsule due to vasomotor disturbance of nutrition may be followed by passage of aqueous fluid through the capsule into the lens substance, producing lens opacity and more or less swelling. If interference of nutrition is arrested before actual disintegration of capsular substance takes place, resolution may follow in the capsule rendering it again non-porous, when lens opacities may under favorable conditions be absorbed; if, however, there is an actual degeneration of the capsular substance, resolution of the lens substance will hardly take place, even after normal nutrition has been reestablished to the lens.

THE SIGNIFICANCE OF THE TRANSPARENCY OF THE RETINAL BLOOD COLUMN

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It is a well-known fact that the retinal blood vessels are transparent and indistinguishable from the retina, but it is not so well known that the blood contained within the vessels is normally opaque. While ophthalmic literature teems with the former fact, it is likewise conspicuous in the absence of the latter fact, although there are numerous references that could be interpreted as confirming or denying the normal condition of the retinal blood column.

Transparency of a blood column means viewing the color of an underlying venous blood column through an overlying arterial blood column, or vice versa. Care must be taken not to place too high a value on this condition when the vessels cross each other on the optic disk, for here the pale color of the nerve head has a tendency to reflect the light more than the retina and thus cause the column to appear more transparent. The length, breadth, tortuosity, and light reflex of the retinal vessels aid us to diagnose changes of the vascular system and other diseases, so also the transparency of the blood column aids us to diagnose blood conditions.

Transparency of the blood column is not a common condition to find in the retina; in fact I have been able to find it twenty times only, in the last 600 cases.

In order to ascertain what diagnostic value we could place on its occurrence, blood examinations were made in all twenty cases. No significant value could be placed on the corpuscular findings, for they varied in direct proportion to the cause of this condition. But it was noted that in all cases the hemoglobin was below normal, in fact it ranged from 30 to 65 per cent. in those cases where the transparency was noticed in blood columns over the retina and 10 per cent. higher in those noticed on the optic disk.

The first ten cases were treated with tonics, principally the syrup of the iodid of iron, and kept under observation until the blood column became opaque. Then a second blood examination

was made which showed that in some cases the white corpuscles had increased and in others the red, but in every case the hemoglobin was above 80 per cent.

Owing to the various ages of the patients, which ranged from 15 years to 65 years, and to the different causes of the anemia, the duration of the treatment varied. In the other ten cases no examination was made of the blood following the treatment, for they were referred to the general practitioner.

Lack of transparency of the blood column does not necessarily mean a normal condition, for we may have a beginning sclerosis of the vessel wall that will obstruct the view of the columns. On the other hand, a transparency when present besides meaning a blood disease means that we cannot have a great amount of vessel sclerosis, for an opacity of the vessel wall means an obstruction to the passage of light waves through the column. Therefore the transparency of the retinal blood column is of diagnostic value in blood diseases as well as in diagnosing arteriosclerosis. We cannot have an arteriosclerosis and a transparency at the same time.

Wherever bluish white streaks mark the vessel wall in the retina and transparent blood columns exist, I believe we can diagnose between a true sclerosis and a transudation of white corpuscles within the lymph spaces of the adventitia, which frequently follow those severe concussions to the eyes in fractured skull and in other causes of retinitis.

Gunn speaks of a transparent blood column caused by the bending of a vein over a sclerosed artery, but this is not due to lack of hemoglobin, as was found in the twenty cases to which this paper refers.

TRANSACTIONS
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THE VALUE OF STEREOSCOPIC RADIOGRAPHS OF THE HEAD

ADDRESS OF VICE-PRESIDENT

J. M. INGERSOLL, A.M., M.D.
CLEVELAND, OHIO

A photograph or an ordinary radiographic plate represents the object viewed in two dimensions only. A stereoscopic picture gives us a reproduction of the object in three dimensions. In other words, we see the object's height and length and depth, just as we see it when we look directly at it. A stereoscopic radiograph enables us to see not only the external conformation of the head, or the chest or any portion of the body, but also to look into the interior of the body and see many of the structures there and their relations to each other.

In order to understand a stereoscopic radiograph, we must learn to look at it with the proper focus. Our skill in interpreting it develops with practice, just the same as we acquire skill in the use of the microscope by practice.

We can all of us secure the necessary skill and knowledge for the correct interpretation of stereoscopic radiographs quite easily, if we will study the radiographs before we operate and then verify our interpretation of them by studying them again after we have exposed the various structures in operating.

All of the accessory cavities of the nose can be seen and quite accurately defined by lateral and frontal stereoscopic radiographs of the head. The size and shape of the maxillary, frontal and sphenoidal sinuses can be distinctly seen and pathological conditions in them can easily be recognized. Often a probe may be passed into these cavities through the normal openings and held in position with adhesive plaster while a radiograph is taken. The probe then gives us a definite point of orientation and helps us in studying the picture.

The differentiation of the individual ethmoidal cells is somewhat more difficult, because they overlap; but it can be done quite definitely with a little careful study.

The orbital cavities show very distinctly in a frontal view and any change in their bony walls due to tumors or other pathological conditions can be clearly seen.

Pressure atrophy in the sella tursica and involvement of the sphenoidal sinuses, caused by tumors of the hypophysis, are very clearly shown in the radiographs.

Stereoscopic radiographs of the mastoid give us very much positive information in regard to the pathological conditions there. The amount of involvement of the mastoid cells can be seen. The position of the sigmoid sinus can be accurately determined, so that we know before beginning an operation whether or not it lies well forward or posteriorly and also its relative depth below the cortex of the skull. Areas of necrosis of the bone over the sinus and the dura can be diagnosed.

The anterior vertical semi-circular canal can almost always be seen; usually the posterior vertical and sometimes the horizontal canal can be outlined. In our earlier work at Lakeside Hospital (Cleveland), we failed to recognize any of the semi-circular canals but we felt sure that they could be seen if we only knew where and how to look for them; so we took a prepared skull in which all three of the canals had been exposed, put some fine wire in the canals and took stereoscopic radiographs of the skull.

The position of the canals was, of course, definitely located by the pieces of wire and after examining these radiographs we studied some of the radiographs of the mastoids which had been previously taken. We were then able to identify one or more of the semi-circular canals in most cases. Now with added experience, we can almost always recognize one and often two of the canals.

We have not yet been able to make a positive diagnosis of brain abscess from the stereoscopic radiographs alone, but we have three plates showing iodoform gauze in brain abscesses which we have operated on. Careful study of the radiographs taken before the operation, and comparison of them with those showing the gauze in the abscess cavity after the operation, brings out certain areas which are, at least, suggestive of brain abscess.

We believe that, as our experience and skill in interpreting such radiographs increases, we will be able to definitely recognize many cases of tumor and abscess of the brain.

All of our radiographs were taken by Dr. Walter C. Hill, Dr. George F. Thomas and Mr. J. E. Olivenbaum. Much credit is due to them for their interest in the work and their valuable suggestions.

THE SOCIOLOGIC ASPECT OF DEAFNESS, CON- GENITAL OR ACQUIRED IN EARLY LIFE

WITH A SUGGESTION FOR A BETTERMENT THROUGH
INDIRECT EFFORT

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BURLINGTON, IOWA

Were it not for the twofold fact that otologists must eventually decide on what constitutes total deafness, and will be looked to more and more to curtail its occurrence, both by prevention and treatment, this topic might be left wholly to the consideration of educators and economists. But the rosy view of the future for this class of unfortunates, painted by enthusiasts among the educators, doubtless encouraged thereto by enthusiasts among the otologists, bids fair to create an expectation on the part of the public, which, especially if it should not be realized, will make the legitimate work of both educators and otologists increasingly difficult.

According to John Dutton Wright¹ there are approximately 50,000 so-called "deaf mutes" in the United States, for whom the great problem is to furnish more effective means for communication with their fellow men, and more remunerative occupations, if they are to have the more intimate relations with society which, in his opinion, is their due. Of paramount importance is the means of communication, and that, also in his opinion, means nothing less than the use of actual speech associated with lip reading. In other words, we must teach the deaf to speak and abandon the sign language. But this presupposes, first, that there can be found a sufficient number of teachers competent to carry on such a work, plus the munificent state to bear the expense; and second, that the deaf will respond in measure commensurate with the effort involved.

Assuming for the present that these teachers and funds will be forthcoming, although this remains to be demonstrated (for it is equivalent to the support of a small army), we must yet inquire about the prospects of response from the beneficiaries.

Of the 13,000 pupils now in the schools for the deaf (again I quote from Wright) and those in attendance for the past ten

1. *Volta Review*: Address before Physicians' Association, New York City.

years, approximately 75 per cent. have had oral instruction; but of these only 25 per cent. have gone beyond the experimental stage, i. e., made practical use of it.

In the light of the estimate that 27 per cent. only of "deaf mutes" are totally deaf, this naturally suggests two questions, viz.: (1) Is this 25 per cent. the intellectual limit of its application? (2) Does it indicate the number of those who, after more or less practice, consider it an improvement on other modes of communication?

In considering the first question we must remember that total deafness means deficient brain capacity in varying grades, from just those things which would come to it through the medium of sound, to those grosser defects, the sequelae of the meningitis which is so often the causative factor. In consequence of this, the 73 per cent. who are supposed to retain "islands of hearing," and therefore more favorable subjects, may not all be eligible. In considering the second question, we must estimate the influence of an artificiality in the process, recognized alike by the producer and receiver. Makuen,² in his contribution to the symposium on "The Deaf Child," makes this statement: "Spontaneous speech development takes place only as the individual is capable of hearing speech sounds, both subjectively and objectively; and speech acquired in any other way is a forced and artificial product." To the deaf person, therefore, who objects to being in the "limelight," and that means most of them, this forced and artificial product will make small appeal; for its first effect, through unusual tone and inflection, is to make the user conspicuous.

Something like twenty years ago, while making with my colleague, Dr. Hobby of Iowa City, a systematic examination of the pupils in the Iowa School for the Deaf, the superintendent's son, himself a pupil, was exhibited as a triumph for oralism. Later, in the privacy of our room, we found ourselves in accord on this impression: "Heartrending! Were it my child I would rather it remain forever silent." And quite recently, in a man from the Philadelphia school, with exclusive oral instruction, I experienced the same shock. When I learned that this man could hear until the age of 7 years, had but recently taken up the sign language, and now spoke with reluctance, I had further confirmation of early impressions. It may be that, in the not distant future, familiarity with this peculiar speech will

2. *Laryngoscope*, June, 1910.

soften or blunt our sensibility to it; but the intelligent deaf person can hardly escape the feeling that he or she will be, at best, just a little less a curiosity than Helen Keller. Incidentally, let me here remind you that of the multitude (a multitude that would doubtless pay willingly for the privilege) who gaze with awe, and mayhap inspiration, on Helen Keller's achievements, few stop to contemplate the patience, perseverance and resourcefulness of her teacher; which is infinitely more wonderful.

The task of bringing any class of defectives up to a reasonable equality with those of unimpaired faculties must always be colossal; not only from the viewpoint of scientific achievement, but as well from the indisposition of the public to render the needed assistance. In this instance the great obstacle in the way of change is the feeling that the duty is already well done. The state furnishes as good a school for the deaf child as for the hearing, and in a material way does more for it, in that it also provides food and lodging—and clothing if necessary. In Iowa such education is compulsory. The idea, too, that the deaf child must have an exclusive oral environment, absolutely barred from the language of signs, involving so much additional expense, may be met with skepticism; and for these reasons: (*a*) Every parent uses signs as a means of teaching the hearing child to speak; (*b*) the good preacher, good orator and good actor (and who of you has never been a devotee at the shrine of Punch and Judy?) is distinguished from the indifferent ones of his class by his ability to press his points with appropriate gestures, and signs often more expressive than words; (*c*) now that the Tower of Babel, with its "confusion of tongues" is again a reality, through the advent of thousands who speak, but not in our language, it has become a necessity to use the language of signs extensively in all the avenues of industry; (*d*) from time immemorial the deaf have been educated by a system of signs—in most schools are so educated to-day, and the majority of those who have acquired speech make more use of the signs; (*e*) in short, when 89,950,000 people are using the sign language, more or less, every day, it is hard to imagine a condition in which the 50,000 scattered broadcast can be shut out from it.

The sign language, therefore, however much it may interfere with the development of oralism, is here to be reckoned with. Nor is it necessarily a relic of barbarism. At the recent congress of the deaf in Paris, a world's congress with educated

people from many lands, the sign language prevailed and was found adequate for a general interchange of ideas. As a matter of fact, everybody knows, or should know without such evidence, that the sign language is the only universal language. It seems, however, to be no less a fact that the general conception of it is wholly inadequate. Even De l'Epee, whose name is inseparably connected with its best known usage, had a singularly narrow view of its merits. Volapuk, which had its brief day, *et id omne genus*, were attempts at a universal language of words, a superhuman, and in view of the possibilities of signs as conveyors of ideas regardless of words, a gratuitous task. De l'Epee and his colleagues seem not to have risen above this weakness. Provision for words gave existence to the manual alphabet.

The real sign language, however, has to do only with subject, object and action, leaving each race to give them such word expression as it will, and may profitably be *taught to the hearing* as well as the deaf. It must also be acquired just as any other language is acquired; and on this account it is best to start with it in childhood before there is definite knowledge of the construction of spoken language. This means, before grammar is taught. Some of us know, better or worse, the French, German, Italian, Spanish, etc., but only those of us who had such environment in childhood can express ourselves in these languages without that awkwardness which imperils our intelligibility to those of the mother tongue. Thus it is probable that none of us would ever become proficient in the sign language; but our children or grandchildren might become facile to a surprising degree, and that without a manual alphabet. For, the manual alphabet need be only an incident, just as we spell out those technical expressions to the stenographer who might not transcribe them correctly. When it is realized that one may think in signs the same as in words—as those skilled in their use really do this—it may even be a matter of surprise that the sign language was ever considered a makeshift, and there were few to do it reverence.

As a basis for that systematic arrangement which is essential for the study of any language, we already have the so-called natural signs which are practically common to all peoples. From these, infinite elaboration is possible; but for the average person a thorough familiarity with these alone may be sufficient. The

English language is composed of many thousand words,³ but the average person with fair education seems to get along with at best a vocabulary not exceeding 2,000; and a single sign may be equivalent to several words—sometimes more expressive.

That many desirable results could accrue in a general acquisition of the sign language seems almost beyond question. For present purposes, however, it is sufficient to point out: (1) that through the element of personal profit in it, its teaching would command public interest and support; (2) that through a common means of communication between the hearing and the deaf child they would be brought into closer relationship; and (3) that through this closer relationship there will naturally follow an appreciation of the blessing in hearing and the curse in deafness, now known only to those under the curse and those besought to lift it—an appreciation which will foster that sympathy so aptly described by Dr John Brown of Edinburgh as the motive and not the emotion.

This argument, while in the main a plea for general instruction in the sign language for the purposes set forth, has also a medical bearing. Were it germane to the subject, I would make some observations on the difficulties attendant on the determination of the degree of deafness in the deaf mute, founded on the examination, physical and functional, of the pupils in the Iowa School for the Deaf, to which I have referred; and how I thought that I discovered that, in those with negligible drum change, the remnants of sound perception were mostly to be found in the left ear. But under the title, matters of policy alone may be appropriately considered.

The brilliant work of Wright and a few others who like him are advocates of oralism pure and simple, gives us, as otologists, a new question to consider when we are consulted about the management of the deaf child. Some of our number have already committed themselves as endorsers of this method for the child who possesses "islands of hearing," especially those within the Bezold scale; and they are men of standing, whose influence may go far with those who have limited association with this class of children. I cannot but think that this endorsement, if allowed to go unchecked, will work misfortune to the otologist and deaf child alike. It is not a question whether the deaf child can be made to speak. Undoubtedly the great majority of them can, some of course much better than others. It is

3. Century Dictionary, 220,000 words added since 1889.

rather a question whether the deaf child's welfare and happiness will thereby be measurably enhanced; and this has not yet been proved. However natural our expectations that oralism would do much that is claimed for it, be worth the added cost, we find, even in the last ten years with its largely increased practice, only sporadic realization of these expectations. It is hardly thinkable that this can be explained on any ground but natural causes, mostly beyond the range of human effort; and on this account it becomes our plain duty to warn the parents of deaf children against too great expectations, pointing out as far as may be the obstacles peculiar to the physical and mental make-up of the individual, and those dependent on social conditions. As explanatory of the latter I know of nothing more comprehensive than the declaration of the lady from whose letter Dr Gallaudet quoted in his contribution to the symposium previously referred to. Every otologist should have a copy of this on his desk.*

For the betterment of this social condition, the general acquisition of the sign language, which I here advocate, may be looked on as speculative. But, from a well-known teacher of the deaf, one who has spent practically his whole life among them, I have the assurance that such a course would be hailed with acclaim by the deaf.

APPENDIX

* Extracts from letter quoted by Dr. Gallaudet: "It seems to me the pure oral teachers expect too much of both the deaf and the hearing. They think that the former should be capable of an equality with the latter, which is physically impossible. They think the hearing should receive the deaf with open arms, or at least meet them half way. They ought to, of course; but the practical question is: Do they? In most cases, No. Where there are deaf friends or relatives, something of interest and kindness will be shown by the hearing; but with ordinary people the deaf are simply strange creatures like the idiotic or insane, though of course in a less degree. The great majority of oralists are absolutely ignorant of the way they are laughed at behind their backs. I myself knew nothing of this while I had home and family to insure me respect, but I've had some bitter experiences since then. For this reason, if for no other, those with bad voices should *not* be forced to talk. They simply make themselves a laughing-stock among the hearing. I have been told that my voice was not specially disagreeable; yet I have known hearing friends to pass me on the street without recognition, and when I demanded an explanation, confess that they did not wish the friends they happened to be with to hear me speak. Is that not enough to seal the lips of any sensitive oralist? In all this I am putting myself in strong antagonism to my school, but it is not to be helped. Truth and common sense should be considered as well as theory, and with the theories of the pure oralists I cannot agree.

"I insist on signs and signs only in public speaking to the deaf. On March 20, 1910, I was present at the confirmation service at Trinity Church, Boston, where Mr. Smearing interpreted to us the sermon of Bishop Lawrence. When I came to read the printed report of that sermon I found nothing new. Had I been seated with the general audience I should not have known a word from beginning to end."

DISCUSSION

DR. OTTO GLOGAU, New York: I was very much astonished to hear the essayist advocate the sign language before an otolaryngological meeting. All over the world the otologists are in favor of the oral education of the deaf, as this is the more scientific method and the only way of preventing the deaf from becoming pariahs of human society. Even in France, where the sign language originated, lip reading and oral education of the deaf are now in vogue. In Germany and Austria, Urbantschitsch, Bezold and Denker showed ways and means of making use of the remnants of hearing met with in most of the deaf, in order to facilitate their instruction in the oral method. The essayist is in favor of that clown language that characterizes the deaf for ever as such and is bound to render his life a miserable one. It was my honor to demonstrate in a lecture on the oral education of the deaf Miss Helen Keller before a large audience. Simultaneously Professor Wright demonstrated some of his orally educated pupils. The results obtained by the oral method are astonishing. A deaf patient of mine is working as a stenographer and typewriter in an office, reading the dictation from the lips of her employer. We frequently meet in society intelligent people with whom we converse very freely, later on we learn that we were enjoying the results of oral education of the deaf. It is up to the American otolaryngologists to protest against the sign language as an unscientific and barbarous method.

THE PRETURBINAL OPERATION ON THE MAXILLARY SINUS

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PHILADELPHIA

In 1908, at the meeting of the American Medical Association in Chicago, Canfield described a new method of operating intranasally on the maxillary sinus. This was termed "The submucous resection of the lateral nasal wall in chronic empyema of the antrum."

This method was based on an entirely new principle, that is, making an extensive resection of the lateral wall of the nose submucously through a comparatively small incision in front of the anterior attachment of the inferior turbinate (Fig. 1). The bony structure of the inferior turbinate was then removed with a corresponding amount of the osseous wall forming the inferior nasal passage. A portion of the crista piriformis, corresponding to the inner part of the anterior antral wall, was then resected and a flap formed from the mucosa of the inferior nasal passage was turned and packed with vaselin gauze onto the antral floor, thus completing the operation (Figs. 2 and 3). By this method not only could the interior of the maxillary sinus be inspected, but a large permanent opening into the nose was created, extending practically the entire length of the inferior turbinate.

The after-treatment consisted in removing the gauze on the fourth or fifth day and touching subsequent granulation tissue with pure nitrate of silver. Complete healing occurred in from ten days to three months.

The author claimed the following advantages for his method: (1) May be used as a substitute for the Caldwell-Luc, as it is quite as radical without having the incision through the mouth; (2) a direct view into the antrum is obtained; (3) the after treatment is simplified and often unnecessary.

During the discussion which followed, a number of exceptions to the method were taken; thus J. W. Murphy thought that removal of so much of the wall was a rather heroic procedure.

Jansen of Berlin condemned the use of the flap, as instead of furthering the growth of epithelium onto the floor of the sinus it actually prevented it and lengthened the time of healing.

Freer regarded the operation as needlessly formidable and elaborate for all but the extremely rare case in which severity is marked. Other speakers condemned the removal of the lower turbinate bone, as the subsequent shrinkage would cause widening of the nasal lumen, thus favoring atrophic rhinitis.

After careful consideration of the different phases of this method one must draw the following conclusions: (1) It is too extensive and involves the loss of tissue which is important for the nose to properly perform its physiological function (bony structure of inferior turbinate); (2) the submucous resection is

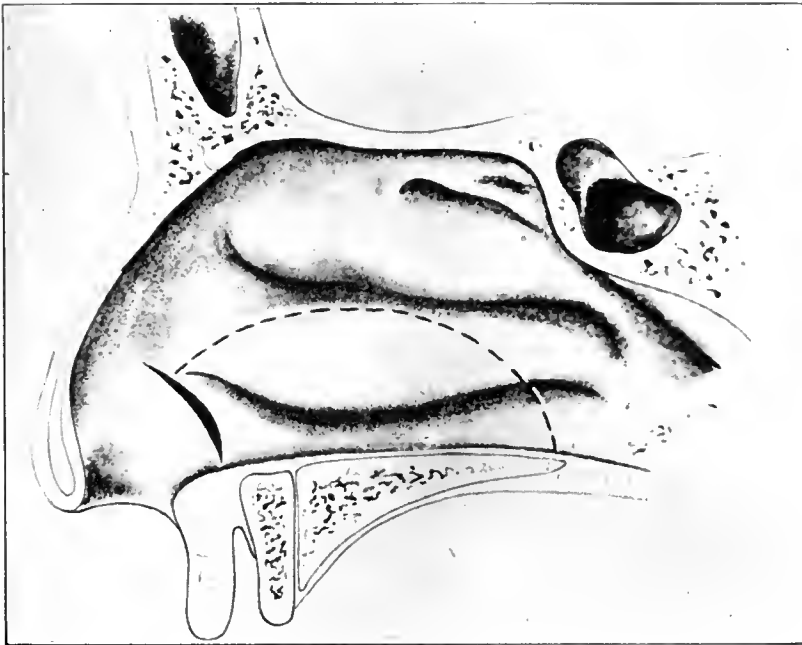


Figure 1.

difficult, tedious and unnecessary; (3) the same results can be obtained by a similar operation of less magnitude.

Sturmamm in 1910 brought out a similar operation but without the submucous resection. His idea was to make the procedure an intranasal Denker operation, that is, the incision was made in the nose instead of in the mouth.

The method was performed in the following manner: The inferior nasal passage and canine fossa on the affected side were painted with a 20 per cent. solution of cocain. A solution of cocain-adrenalin was then injected under the periosteum on both the facial and nasal side of the crista piriformis. After anes-

thesia was complete an incision was made in front of the inferior turbinate and the crista piriformis exposed. Submucous resection was made on the nasal side, subperiosteal resection on the facial. The crista piriformis was then removed with bone forceps, drills, etc., and as much of the facial and nasal bony walls as was necessary in order to directly inspect all parts of the sinus cavity including the roof. The sinus was cleansed with tampons saturated in peroxid of hydrogen, and a mucous membrane flap made from the lateral nasal wall directly behind the

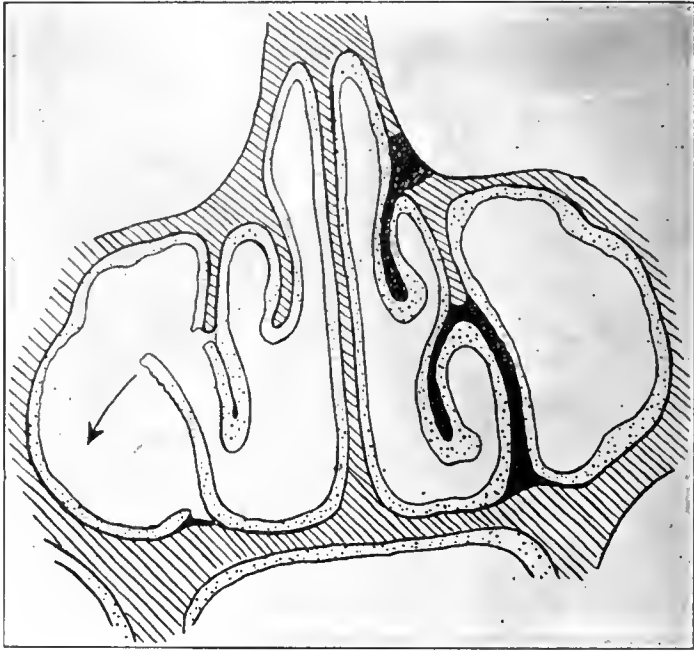


Figure 2.

incision and turned backward into the sinus and along its floor. This flap was held in position with a tight tampon of iodoform gauze, which was allowed to remain five days. Tampons were then changed every two days for two weeks, then omitted. Complete healing occurred in from four to seven weeks.

These operations, while presenting a great advancement in surgical technic, nevertheless left something to be desired, the first (Canfield's) being too difficult and extensive, the second (Sturmann's) being over-radical by removing too much of the bony structure as well as several minor defects (retention of flap, etc.). This form of operation, however, always appealed

strongly to me, and during the application of these methods the following technic seemed to take its place through a series of gradual evolutions until it has now become, in our department, the standard intranasal operation on the maxillary sinus, whenever an intranasal procedure is indicated. It has always been done under local anesthesia although general may just as readily be employed:

1. The nasal cavities on both sides are cleansed by douches of warm normal saline solution.

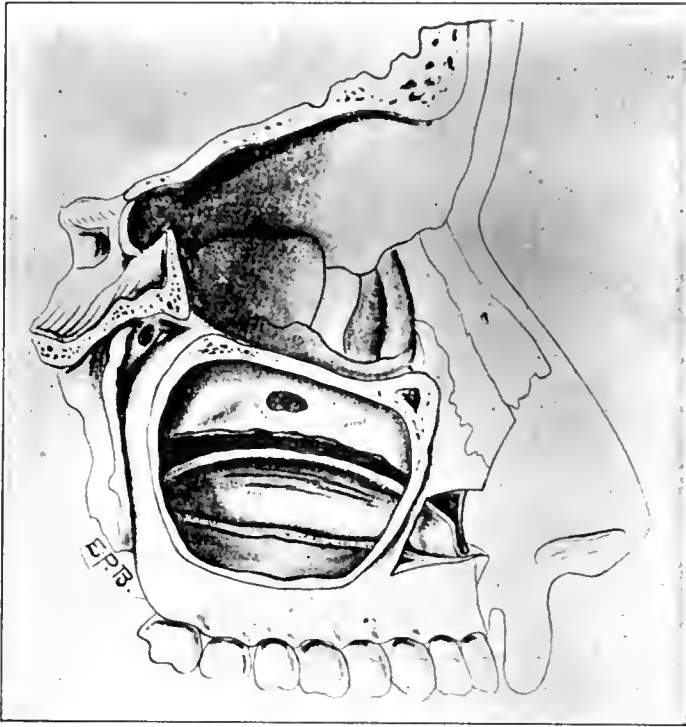


Figure 3.

2. The entrance to the nose including that portion immediately in front of the anterior attachment of the inferior turbinate, both above and below, is anesthetized by painting with a solution of cocain 20 per cent., to which one-fifth its volume of adrenalin chlorid has been added.

3. When the anesthetization is complete a solution containing novocain 1 per cent., adrenalin chlorid 1:10,000, peptone pure 10 per cent., normal salt solution 89 per cent. is injected beneath the mucosa on the nasal side of the piriform aperture

and subperiosteally on the facial side of the same structure, so that all that region around the anterior attachment of the inferior turbinate as well as the inner portion of the canine fossa wall will be desensitized. It will not be necessary to use much over 5 or 6 c.c. (80 to 100 gtts.) (Fig. 4).

4. After waiting about ten minutes a perpendicular incision is made slightly in front of and above the anterior end of the inferior turbinate, extending well down into the floor of the nose. This incision should sever all tissues down to the bone. A



Figure 4.

second incision is made directly back of this, meeting the first one above and below so as to excise a spindle-shaped piece of mucous membrane (Fig. 5).

5. After controlling the hemorrhage with adrenalin tampons, a small elevator is used to elevate the periosteum from the crista piriformis both externally toward the canine fossa and internally toward the inferior turbinate until a sufficient portion of the bone is exposed (Fig. 6).

6. The antrum is now attacked with a chisel having a concave surface, by applying it to the crista piriformis first above, then below, removing the loosened bone with a pair of strong forceps. While the antrum may be opened by continuing this method, it is better to substitute an electric trephine, as a smooth, round opening is thus obtained, insuring against spicules of bone being driven into the sinus.

7. Enlarge the opening to any desired size by means of the ordinary curved frontal-sinus rasps.



Figure 5.

8. Flush out sinus and after drying pack a thin strip of gauze saturated in the cocain-adrenalin solution and allow it to remain five minutes. This not only anesthetizes the mucosa, but also by its hemostatic action clears the cavity of blood and permits a much more satisfactory inspection of the interior (Fig. 7).

9. Introduce an ordinary hard-rubber ear-speculum into the opening and thoroughly inspect the antrum for polypoid degenerated mucosa, areas of granulation tissue, necrotic spaces, etc. This procedure is readily accomplished if sufficient bone has

been removed. The nasopharyngoscope is used for inspection of the roof, lacrimal region and ostium.

10. Introduce a curet and remove all portions of diseased and degenerated mucosa, not overlooking the floor, posterior-inferior and anterosuperior angles; the latter can only be reached by a right-angle curet.

11. Again inspect the interior of the sinus, using cotton or gauze pledgets dipped in pure adrenalin chlorid when necessary for cleansing purposes. If all polypoid tissue seems to have been removed and the cavity seems clean, again irrigate, and after allowing the fluid to run out, pack loosely with iodoform gauze.

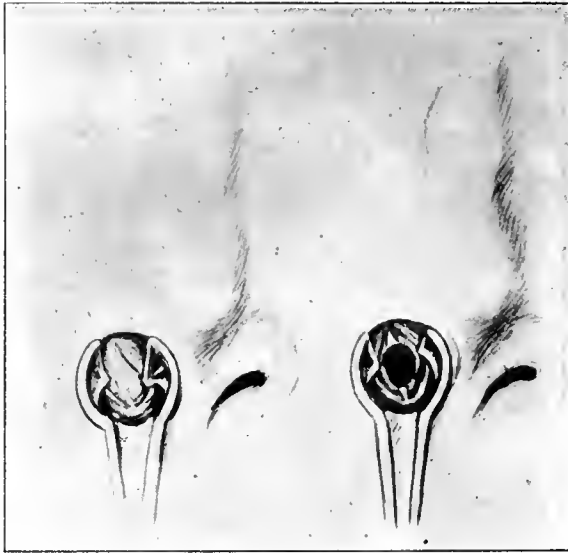


Figure 6.

Figure 7.

After-treatment: The gauze is removed in fifty-eight to seventy-two hours, depending on the amount of secretion; if it remains moderately dry it can be permitted to remain even as long as one week. After its removal, the cavity should be cleansed by irrigation and lightly repacked with iodoform gauze. The treatments are continued every second day for ten days to two weeks, when the packing can be permanently discontinued. It is remarkable how quickly the discharge lessens after the packing is abandoned.

The treatments (irrigation and insufflation) are continued at increasing intervals for about four weeks, when, in ordinary cases, the patient is discharged cured. Certain cases, however,

in which the permanent pathological changes in the mucosa had necessitated the removal of large areas of the lining membrane and in which one of the forms of the external radical operation was indicated, require longer and more energetic after-treatment to promote granulation and cicatrization of the already enfeebled tissues. In these cases the discharge continues with little change after the packing has been discontinued. After thorough cleansing and drying, 1 ounce of a 25 per cent. solution of nitrate of silver is slowly injected and allowed to remain five minutes, the patient bending the head toward the shoulder of the affected side. This is continued every second day, the silver solution being used in increasing strength (to 75 per cent.), if the weaker solutions do not appear to be sufficiently stimulating. It is surprising how little discomfort these injections cause, practically no more than the like quantity of sterile water.

Precise information regarding the healing and general condition of the sinus interior can always be obtained by means of the nasopharyngoscope and I know of no condition in which the use of this little instrument gives more satisfaction than after this operation. Small areas, such as necrotic spots and suppurating foci, which refuse to heal are easily located and directly treated with pure silver nitrate, then reinspected to ascertain whether all parts have been touched. In this manner final and definite healing is brought about. During the after-treatment, it is necessary to always keep the artificial opening of the sinus in mind, as it shows a marked tendency to close with astonishing rapidity. This can be prevented by occasionally curetting the edges and applying a caustic. While the gauze packing is in place this will not occur and indeed this is one of the reasons for the several repackings. After healing has been established, this opening will gradually close until that side of the nose is to all intents and purposes quite as normal in appearance as the opposite side.

Advantages over other intranasal procedures: (1) The sinus can always be inspected either directly or through the nasopharyngoscope, and the progress of healing noted; (2) the drainage is at the lowest and most accessible point reached through the nose; (3) local applications directly under vision can be made to diseased areas which have proved resisting to treatment; (4) the inferior turbinate is not only preserved in its entity, but remains uninjured; (5) the operation is practically painless, if the anesthetization is properly carried out; (6) the period of healing is considerably shortened and the number of after-treatments greatly decreased.

FURTHER OBSERVATIONS ON THE PHYSIOLOGY
OF CONCENTRATED COCAIN-ADRENALIN SOLU-
TIONS FOR INDUCING LOCAL ANESTHESIA
AND TECHNIC OF APPLICATION IN EYE,
EAR, NOSE AND THROAT SURGERY

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The nasopharyngeal mucous membranes are notable for their great vascularity and rich nerve supply, and this structural arrangement enhances their functions of absorption and secretion and renders them especially responsive to stimulants or sedatives brought in direct contact with their surfaces.

The accessibility of these surfaces for the direct application of drugs to induce local anesthesia has led to much investigation and experimentation to discover an agent that is effective and safe.

The character of the muscle fibers composing the blood- and lymph-vessels of the mucous membranes has a marked bearing on the physiology of local anesthesia. You are cognizant, of course, of the fact that contractility is the characteristic property of muscle substance and that muscles and muscle fibers are divided into two great classes, the striated and non-striated, determined by their structure and the functions they perform. The muscles that control the extremities are striated and distinguished by their rapidity of motion. The muscles composing the walls of the blood-vessels are non-striated and are sluggish in movement.

Irritability is a second vital property of muscle and nerve protoplasm, and while it finds its best form of expression in the nerve substance, the muscles are hardly less susceptible to stimulants or irritants.

Conductivity, or the property which enables a substance when stimulated at one point to transmit the impulse or impression to remote regions, is possessed in the highest degree by the nervous system. Thus we note that different tissues manifest divergent functions, and, without question, structural peculiarities and specialized functions are intimately related. Striated muscle fiber, for example, responds quickly to stimulation and

non-striated muscles (the type controlling the blood-vessels) respond more or less slowly, while nerve fibers are remarkable for their conductivity.

On these vital properties and characteristics of muscle and nerve function is based the rationale of the efficiency and safety of the concentrated cocain-adrenalin solutions in local anesthesia.

As indicated above, the marked vascularity of the nasopharyngeal mucous membranes renders them remarkably absorptive, therefore very responsive to the action of drugs brought in direct contact with their surfaces. While this unusual absorptive function facilitates and enhances the effect of drugs applied to induce local anesthesia, it is obvious that if the drugs are toxic it exposes the system to general intoxication, unless the absorption is limited for the most part to the superficial or local circulation. Concentrated adrenalin solutions do this, and for the following reasons:

1. Strong cocain-adrenalin solution is a most powerful stimulant to the sluggish unstriated vasoconstrictor muscle fibers of the blood- and lymph-vessels; the degree, rapidity and duration of this constriction being relative to the strength of the drug solution employed. Therefore the more concentrated the cocain-adrenalin solution the more quickly and completely the local circulation is blocked, absorption into the general circulation is prevented and toxic effect of the drug is obviated. Moreover, by restricting the cocain to the superficial tissues, the drug comes in direct contact with the nerve endings and nerve fibers, is retained there, and of course the degree of anesthesia is in proportion to the strength of the solution. When the drug comes in contact with nerve trunks, it may follow the axis cylinders of the nerve fibers distally or proximally and be relayed through nerve cells and ganglions to the neighboring or even remote areas, producing anesthesia, which phenomenon is designated as nerve-trunk anesthesia, or "blocking." Reference will be made to the technic of this process further on.

2. Leshure¹ in a timely thesis on this subject some time since called attention to the fact that fluids of high density, such as concentrated cocain solutions, are not readily taken up by the blood-vessels. The laws he cites "governing the absorption of aqueous drug solutions are: 1. A fluid passes through a membrane with a rapidity inversely proportional to the density

¹ Leshure, John: The Physiological Action of Strong Cocain-Adrenalin Solutions, New York Med. Jour., Feb. 6, 1909.

of the fluid. 2. The rate of absorption varies directly with the fulness and tensivity of the blood-vessels and lymphatics. 3. The slower the movement of the blood- and lymph-streams, the slower will be the rate of absorption of the fluid.

"These well-recognized laws of physiology explain the local retention in the tissues of the strong cocain-adrenalin solution, and the lasting anesthesia and ischemia following its use."

According to this first law, the very concentrated cocain-adrenalin solution (90 per cent.), which has a specific gravity of 1.180, is absorbed much more slowly than 10 and 20 per cent. solutions of 1.020 and 1.040 specific gravity, which are nearer the specific gravity of blood-serum, which ranges from 1.025 to 1.030. The great density of the strong cocain-adrenalin solution prevents its ready absorption and it is limited to the superficial tissues; but as both drugs powerfully contract the blood-vessels and lymph-vessels, the second and third laws enumerated above are made effective as the fulness and tension of these vessels are lessened and the movement of the blood- and lymph-streams slowed.

Basing my argument on the above sound physiological laws, my contention is that the degree, rapidity and safety of cocain-adrenalin solutions in producing surface anesthesia are in proportion to their strength. And my experience with different strengths of solutions convinces me more and more that the greater the concentration the greater their efficiency, and the less their toxicity: hence my urgent plea for their use.

TECHNIC

In a previous communication² on this subject, I detailed the technic of surface anesthesia with cocain-adrenalin solutions in ear, nose and throat and eye surgery. At present I wish to confine myself to the discussion of a method of inducing nerve-trunk anesthesia of the tonsils.

Some four years since Sluder³ did pioneer work in nerve-trunk anesthesia by surface cocainization of Meckel's ganglion and the internal nasal nerve for all surgery of the lateral nasal wall and postnasal regions. You are aware that the cephalic portion of the sympathetic is composed of four small ganglia connected with the three divisions of the fifth nerve, connected

3. Sluder, Greenfield: Syndrome of Sphenopalatine Ganglion Neurosis, *Am. Jour. Med. Sc.*, December, 1910.

2. Davis, George E.: A Safe and Efficient Method of Local Anesthesia in Ear, Nose and Throat, and Eye Surgery, *The Post-Graduate*, August, 1913.

with each other and connected with the cervical portion of the sympathetic. With these intimate and direct connections, we can understand how an anesthetic applied to any of these ganglia or their branches may be conveyed to the others or to the several organs or regions they supply.

The sphenopalatine, or Meckel's ganglion, is the largest of these four cranial ganglia, situated in the sphenomaxillary fossa on the lateral pharyngeal wall just posterior to the middle turbinate. Its descending branches, in conjunction with the tonsillar branches of the glossopharyngeal, form a plexus around the tonsil and supply the tonsil and the muscles embracing it, to wit, the palatoglossus anteriorly, the palatopharyngeus posteriorly and the superior constrictor externally.

My present practice for anesthetizing the tonsils is by applying a saturated cocain-adrenalin solution on a cotton-wound applicator, adjusted to the lateral nasal and pharyngeal walls through the inferior nasal fossa, contact being made beneath the posterior tip of the inferior turbinate, extending posteriorly to a point in line with the eustachian orifice. By this procedure the nerve trunks of the descending palatine branches of Meckel's ganglion, which supply the tonsils, are completely anesthetized and sensation soon blocked. Liberal use is made of the solution, as much as 5 to 10 minims, but the pledget of cotton on the applicator must be of sufficient size to hold the amount of the solution and to prevent it from spreading. To obviate the flow of mucus from carrying the cocain into the throat, a second applicator wound with a much larger pledget of cotton, is adjusted immediately beneath the applicator holding the cocain solution. These applicators are allowed to remain *in situ* for fifteen or twenty minutes. In the meantime careful applications of more of the anesthetic may be made directly to the tonsil and its pillars.

By this method, in the great majority of cases, perfect anesthesia and practically complete ischemia may be induced in the tonsils and neighborhood tissues. Children are not suitable cases for cocain anesthesia.

A NEW SUBMUCOUS SEPTAL OPERATION

OLIVER TYDINGS, M.D.

CHICAGO

This operation was first performed on a girl 9 years old in 1909, for the correction of a nasal deformity due to trauma, received some years before.

The triangular cartilage had been fractured and it had united at nearly a vertical angle, the anterior edge projecting into the right nasal cavity, the apex into the left, producing marked stenosis on each side. And, while in that case it was limited to the cartilage subsequent work done by myself and later by my associate, Dr. O. J. Nothenberg, has demonstrated that it has a wider range of application than I at first thought.

The advantages are: First, it preserves intact all the structures of the septum, unless there should be a redundancy of tissue, in which case, to that extent, it should be removed.

Second, when correcting a displacement of the so-called columnar cartilage, any operation which removes this part of the septum leaves a lack of sense of resistance while wiping or touching that part of the nose, which is somewhat disquieting to the patient.

Third, where there is a distinct dip to the tip of the nose, this can be elevated making quite an improvement in the appearance.

FORMS OF DEFLECTION

The three most frequent are: First, the purely cartilaginous, in which the displacement is anterior to the mesethmoid and vomer articulation.

Second, those beyond this articulation.

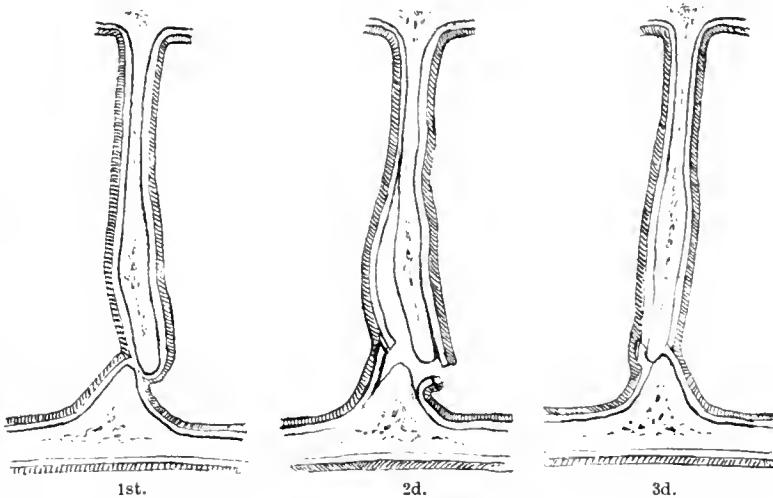
Third, those which involve both the cartilage and bone.

CARTILAGINOUS

In the pure cartilaginous deflections or displacement, whether due to trauma or developmental causes, the incision is made on the convex side just anterior to the deflection going through the coverings and cartilage to the perichondrium on the concave side, then taking a slightly curved elevator, with its concavity towards the concave side of the septum, you elevate the peri-

chondrium and its overlying membrane past its juncture with the mesethmoid articulation, moving along the line of least resistance, which is usually near the center of the cartilage going beyond the mesethmoid and vomer.

When you have passed this articulation you elevate toward the floor, in the line of least resistance, until you have reached it, then with Freer's angular dull elevator (Fig. 1, *B* of his series) you start forward, hugging the vomer until you reach the interlacing periosteum at the superior-anterior border of the vomer, which you sever with Freer's sharp angular elevator, Fig. 1, *A*; now proceed forward until you have elevated the perichondrium on the concave side of the cartilage, which will



frequently take you into the opposite nares to the point of entrance, elevating the perichondrium ahead of you, loosening up all constricting bands until you have the maxillary crest on the concave side laid bare. You then go to the convex side and elevate the perichondrium on that side for about $\frac{3}{8}$ to $\frac{1}{2}$ inch along the border to form a flap, when you have placed the cartilage in the perpendicular position. After this you release your cartilage by severing all adhesive bands and push it over in the groove you have made along the maxillary crest on the opposite side. You then place in position your flap, which usually drops there, and introduce your retaining splint. For this purpose I have used Ashe's or Kyle's splint, but I early learned that an ordinary shawl pin 2 inches long, with head covered with sealing wax, is the best for this work. This is introduced

through the loose connective tissue just anterior to the cartilage, starting from the concave side, passing through; then turning the pin a little more than parallel to the septum, you reenter the mucosa and try to get under the perichondrium and force it at an angle of about 45 degrees toward the floor of the nose, so as to engage it in the dense structures at the anterior part of vomer and find anchorage in the bony floor. The pin is left in position from six to ten days.

Where you have a marked bending of the septum, it is sometimes necessary after crossing over this elevation to again pass through the cartilage at its juncture with the vomer and find anchorage on the side of the vomer from which you started.

Where you have the so-called columnar displacement, you press the mobile septum and mucosa toward the concave side, making your incision just over the anterior part of the cartilage so as to throw your line of incision, when pressure is released, well back of the tip of the cartilage.

When your work is done, this places it where it will be held in position with the pin, and will also enable you to make a space anterior to the bony septum to receive the cartilage. I sometimes anchor this with sutures, using no pins, and again I use both pin and sutures.

Deflections beginning behind the triangular cartilage and mesethmoid vomer articulation are of two kinds and are due to developmental causes.

In one kind the septum is perfectly straight on one side and protrudes into the other side of the nares. This is due to the squeezing out of the vomeronasal cartilage and can be remedied by elevating the periosteum over this structure and shelling it out of its bed. Generally it is easy of accomplishment. You then replace coverings and introduce a light gauze splint for twenty-four hours.

In other cases both bone and vomeronasal cartilage are deflected. In these cases you start high up on the septum, pressing through the triangular cartilage just anterior to its articulation with the mesethmoid to the concave side and elevate the periosteum over the concave surface, to the end of the deflection, then go to the floor and come forward, severing the interlacing periosteum over the anterior-superior border of the vomer. Now elevate the periosteum over the convex side of the bony septum, the vomeronasal cartilage, remove the cartilage and if necessary sever the bone toward the floor where the bend starts and shove it into position. The bone here is generally very

friable, but more frequently than not you can get anchorage of your part, loosened up on the concave side of the vomer sufficient to hold the parts in place. When you do this you can remove the gauze packing, which you introduce on the convex side, after the part has been held in position for twenty-four to forty-eight hours.

Often you will find the vomer perfectly straight and only the mesethmoid and vomeronasal cartilage deflected, but you must free with a chisel all of the deflected surface before attempting to replace.

In correcting those cases in which triangular cartilage, mesethmoid and vomer are deflected, you start as you would if cartilage alone was involved and your dissection is in the same line, but more extensive. You go just beyond the limit of bony deflection on the concave side, elevating the perichondrium and periosteum to the extreme limit of the deflection. You then return to the side of your primary incision, on the convex side, and elevate the overlying structure, exposing cartilage and bone. You then sever all restraining adhesions, whether fibrous, cartilage or bone, always keeping in mind the interlacing periosteum at the anterior superior part of the vomer and that bone and cartilage have their own separate coverings.

In severing the bone you start at the beginning of the deflection below. You use the chisel and this is more easily accomplished after the removal of the vomeronasal cartilage. After all is free, you place the structures in a perpendicular position, making sure that your soft tissues on the convex side are not pinched by the replaced structures, but can be used as a flap. Then introduce your splints, which include pin and gauze, to retain in position.

You can correct most deflections you will meet by this method, leaving only a small proportion of the thickened and irregular deflections for the classic operation of Professor Freer, to whose inventive genius I am indebted for the instruments which have made this operation possible.

THE DYNAMICS OF NASAL DEVELOPMENT; ITS BEARING ON RESECTION OF THE SEPTUM

WILLIAM WESLEY CARTER, A.M., M.D., F.A.C.S.
NEW YORK

The importance of the nasal septum as a factor in the development of the nose and the part it plays subsequently as an important integral part of the nasal arch have been impressed on me in the course of my work on the deformities of the nose. It is a subject, too, that has been largely neglected, though the importance of its bearing on the submucous operation is recognized at once. That this function of the septum is real and of great practical importance to the surgeon, has been demonstrated to me by the several cases of depressed deformity of the nose, due directly or indirectly to the submucous operation, which have been referred to me for correction during the past few years. Such results are to a large extent avoidable; and I believe a thorough understanding of the force employed by nature in the development of the flattened nose of the infant into the prominent, shapely organ of the adult, will contribute largely to the prevention of such deplorable accidents, which reflect undeserved discredit on one of the most artistic and useful operations in rhinology. In no operation do manual dexterity and experience count for more, and in no procedure do uniformly excellent results testify more eloquently to the skill of the surgeon.

When we consider the various forces that enter into the development of the nose, we can but marvel that the task imposed on Nature should ever result in the development of a symmetrical organ, and one bearing a correct proportion to its facial environment.

Each side of the nose constitutes practically a dynamic entity, and yet these two systems must develop uniformly and almost independently side by side to produce a single organ, on the perfect, symmetrical development of which the comeliness of its possessor depends to a far greater extent than to any other factor.

Owing to the fact that the nose is a protuberance, springing from a more or less flat surface, and receiving its support, its

impulse of growth and expansion from its base only, the poise of its constituent parts is all the more delicate and sensitive to disturbing influences.

The scope of this paper is not intended to include embryology; suffice it therefore to remind you that in the early fetus there is only one nasal cavity; and this does not exist as a separate chamber, but opens directly into the mouth. The division of this by the septum nasi commences before its separation from the mouth is effected by the palatal processes of the superior maxillae and the horizontal plates of the palate bones. The fusion in the middle line of these three segments, i. e., the two palatal processes and the vomer, begins forward at about the eighth week and proceeds backward, and is completed about the tenth week. At this tripartite suture, if the union between the palatal processes is delayed or insecure, or if the lower edge of the vomer reaches this point of junction first, it may push down into the roof of the mouth and cause the ridge of bone frequently noticed in the midline of the hard palate. This ridge may also be caused later in life by the vertical pressure to which the septum is subjected during the formative period of the nose. The development of the flat infantile nose into the adult type is effected chiefly by forces acting from three separate and distinct sources, the two nasal processes of the superior maxillae and the septum. To a disproportion in the action of these three forces are due the variations in the shape and contour of the nose which constitute so-called family and racial characteristics. To this may also be attributed the non-traumatic nasal deformities. The latter may be caused also by the unequal development of the two sides of the nose, especially the two primitive plates from which the vomer is developed.

Deflections of the septum at birth are unknown, in fact they are almost never seen until the forces which take part in the development of the nose become active, which is between the fifth and seventh years. From this time on, until puberty, there is every evidence of the activity of these forces. The most active period of all is during the second dentition, when the growth of the upper jaw is very active. At this time, most of the non-traumatic deviations of the septum begin, as the septum is under considerable vertical tension, and the slightest force, such as the insertion of the finger into the nostril may throw the septum out of the vertical, and being unable to recover its normal position, a deflection in time results.

Normal, symmetrical development may be interfered with by many causes. The most frequent is adenoid tissue, which causes the upper jaw to be undersized and the arch of the palate to be raised. Under these circumstances, the vertical space is too short to accommodate the normal increase in the length of the septum and a deflection results.

The septum, which we will assume is the chief factor in the development of the nasal prominence, is locked in by the bony walls of the nasal cavity. Its vertical growth is opposed below by the arch of the hard palate, and above not only is it opposed, but it is being constantly encroached on by the downward expansion of the base of the skull. Due to the rapid growth of the child's brain, this influence is more pronounced in the highly civilized races, and corresponds exactly with our findings in regard to the frequency of septal deviations. These never occur in the lower animals, and they are extremely rare in the Mongolian and negro races. Personally I have never seen a non-traumatic deviated septum in a negro, and the experience of several men with whom I have corresponded in regard to this matter, who have large practices in the South, corresponds with my own. In these races the nose is broad and flat, and the septum, in addition to being comparatively free from the encroachment of the brain-box, does not seem to have, as in the Anglo-Saxon, the inherent power of growth necessary to raise the bridge of the nose. Certainly it is practically immune to those influences, which, according to McKenzie and others, produce deviated septa in 77 per cent. of the Anglo-Saxon race.

The ossification of the various bones that enter into the formation of the nose begins at or about the same time, the sixth or seventh week of fetal life. The vomer, however, does not begin to ossify until the eighth week. This may add somewhat to the disadvantage which its anatomical position imposes on it, but the relative time at which ossification occurs in these bones does not aid us materially in our consideration of this subject, and we will turn therefore to the structure and position of some of the more important bones and to the general architectural construction of the nose.

In the opinion of the writer, the septum has a most important function in the development of the nasal prominence. Its upper edge, where it lies between the lateral cartilages and between the nasal bones, constitutes the keystone of the nasal arch. But its chief function is that of raising the bridge of the

nose; and this, I believe, is accomplished mainly by the vomer. I will, therefore consider this bone more in detail.

The vomer is placed like a wedge, securely between the body of the sphenoid and the arch of the hard palate. Its anterior border forms an inclined plane, the apex of which reaches as far forward as the incisor crest. Its position is very secure and it is well adapted to serve as a basis from which the force developing the nose may spring. Furthermore, the anterior border of the vomer is parallel with the prospective contour of the nose. The growing vomer therefore acts at right angles on the vertical plate of the ethmoid and the septal cartilage, and the growth of the latter in a downward and backward direction is well resisted by the vomer. The division of the original nasal cavity is effected by the vomerine cartilage, the anterior part of this remaining as the septal cartilage, but the posterior part *not* becoming the vomer. Ossification in the vomer does not occur in cartilage, but it begins in two ossific centers situated in the lower, back part of the membrane which covers the vomerine cartilage on either side. From these centers, which make their appearance about the end of the second month, ossification proceeds forward and upward. The fusion of these two lamellae occurs behind and below about the third month, and their fusion gradually extends forward, absorbing and, to a certain extent, pushing forward the central cartilage and the vertical plate of the ethmoid. This process continues until puberty, at which time the laminae of the vomer have become completely united, and all that remains of the original cleft is the groove on its anterior surface, in which is lodged the septal cartilage. The manner in which this cleft is closed, beginning posteriorly and gradually extending forward, and this process continuing during the years of active nasal development, shows conclusively the importance of the vomer in the dynamics of the nose. On the other hand, if through disease, such as syphilis or atrophic rhinitis, the vomer is destroyed, or if its development is interfered with in early life, a saddle-back deformity, more or less pronounced, develops.

My studies have been conducted more with a view to solving the dynamics of the septum than of any other part of the nose, because of its practical bearing on the submucous operation, and the influence which it has in the production of nasal deformities.

CONCLUSION

The normal position of the upper edge of the septum, which constitutes the key-stone, is necessary in order to maintain the integrity of the nasal arch. In removing the septum, therefore, no instrument should be used that necessitates tugging. For if this upper edge of the septum is removed or displaced, a depressed deformity will result, which can be corrected only by the transplantation of bone. As I first suggested several years ago, a punch forceps is the best instrument for removing the septum. This not only requires no tugging, but it has the additional advantage of enabling one to remove the exact amount of tissue necessary to relieve the obstruction. I have concluded further that the force exerted by the septum is indispensable to the development of a symmetrical nose, and therefore the septum cannot be extensively removed with safety during the years of active nasal development. Fourteen years of age I regard as the limit of safety. On the other hand, the framework of the nose, conforming as it does to the definition of the arch, does not require any external support other than at its two extremities. Therefore the septum, if properly removed after the nose has acquired the correct proportions in adult life, causes no injury from an aesthetic point of view and the architectural strength of the nasal arch remains unimpaired.

DISCUSSION

DR. T. E. CARMODY, Denver: The point Dr. Heitger brought up is very important, but gives the wrong idea; the fact is, that in normal breathers the tongue gives you a negative pressure as it has a tendency to draw the palate down, while in mouth breathers, we have a positive pressure in the mouth and a negative pressure in the nose. Furthermore though the tongue is not held in its normal position between the teeth, laterally, so that both upper and lower teeth fall inward, thus narrowing the arch, while the tip of the tongue pushes the anterior teeth forward.

A PLEA FOR THE ELECTRICALLY DRIVEN BURR IN BONE SURGERY OF THE HEAD

JOSEPH C. BECK, M.D.
CHICAGO

Historical.—The electrically driven burr was first employed by mechanics in woodwork and subsequently adopted by general surgeons in smoothing off rough edges in bone surgery. The dental profession was next to adopt the burr and is still employing it as a routine measure in drilling cavities and taking off portions of teeth. The general surgeon next adopted the burr in opening the skull, and finally the otological surgeon employed it in mastoid work. Jansen was probably the first to use the electric driven burr in mastoid surgery, and Allport in this country was probably the first universally to employ the burr and claim great advantages for it. Pynchon was one of the first to recommend the electric trephine in operations on the septum, in tunneling out a ridge, and Gradle adopted the method about the same time for the same purpose, also recommending it for removing part of the hypertrophied inferior turbinated body. Brophy and many other oral surgeons advised to open the antrum of Highmore by way of the canine fossa or alveolus by means of the drill. Cryer has perfected an instrument and a method for the same purpose that is perfection itself. The antrum has been opened first by means of the electrically driven burr or trephine by way of the inferior meatus by Goodwilly and Friedberg. Halle and Ingalls first employed the electric drill in frontal-sinus operation by intranasal route.

There are no doubt many men that are expert in the use of the electric burr, especially about the mastoid as, e. g., Boetcher and Iglauer; but as a general proposition the method is not very enthusiastically taken up.

Owing to the fact that I have had considerable experience in the use of the electrically driven burr in the past ten years and because I find that so comparatively few otolaryngologists employ this method, I wish to bring this matter before this society in the form of a plea, and hope that it may result in stimulating its trial and adoption, because it merits such a course.

The advantages that this method possesses over other methods, as, e. g., chisels, gouges, curets, punches, etc., are so great and many that I am actually surprised why so many operators persist in their use, even though they may be very expert with these instruments. One of the most important advantages is the completeness with which the bone may be taken away right down to the dura, sinus, nerve sheath, mucous membrane or periosteum, without the slightest injury to these structures. One can actually take down the bony walls of the cochlea and semicircular canals without entering their lumen, and I have dissected the entire course of the facial nerve on the cadaver, without the slightest injury of its sheath.

In order to determine the safety of the use of the burr in the complete removal of a bony structure, I have operated on the cadaver as well as on the living a great many times and have found that the dura will be pushed away rather than be injured. I wish to demonstrate some of the primary sketches (illustration) of the various operations on the mastoid and labyrinth that I have performed, and they, with operations on other parts of the head, will appear in their completeness some future day in a publication, this being a preliminary report.

APPLICATION

I have employed the burr in the following conditions and shall mention only the salient points so far as they refer to the burr:

1. In the simple mastoid, radically performed, every cell, be it ever so small, is opened up and cleaned out, yet the antrum is left untouched.

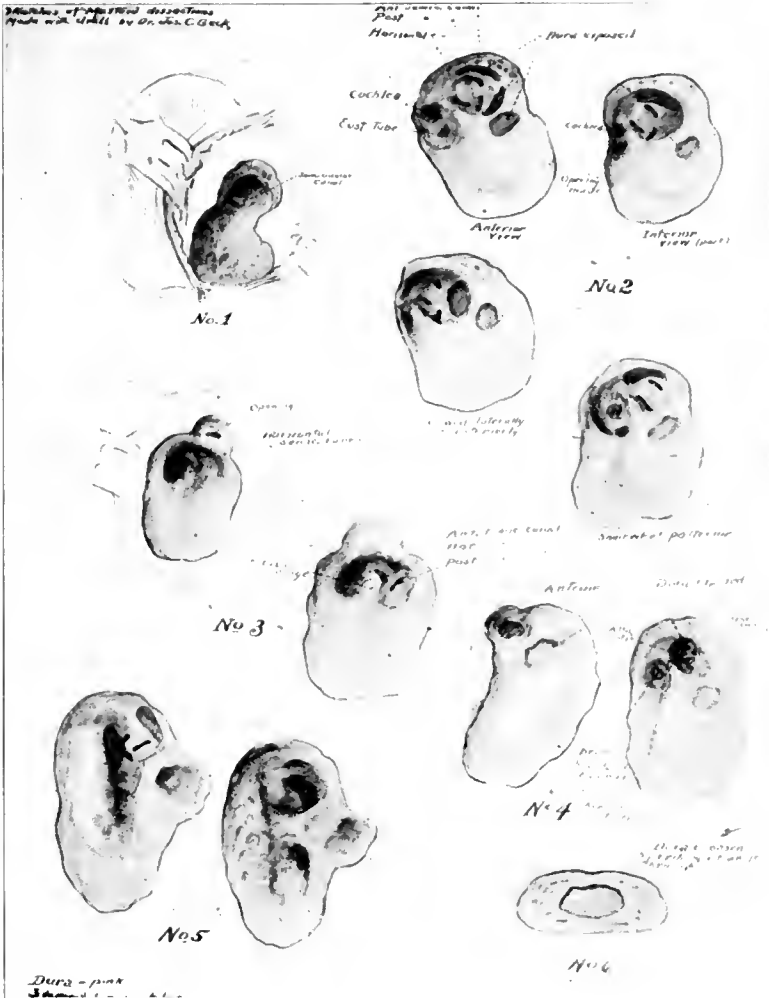
2. In the so-called Heath operation, the points which the author (Heath) lays great stress upon are very well carried out with the burr, especially the close proximity to the annulus, which one can safely approach when removing the posterior wall of the canal.

3. In the so-called Bondy and modern Stacke operations, one can go still farther than in the Heath operation, namely, open the attic without disturbing the ossicular chain.

4. In the typical radical mastoid operation, the perfect removal of the bone over the facial nerve, the thorough removal of the hypotympanum, stand out as great points of advantage, especially as the bone is usually sclerosed. The bony cavity is also made much smoother for immediate skin graft.

5. The labyrinth can be almost completely removed to the internal auditory meatus without having to go outside of the temporal bone.

6. The lateral sinus can be partially or completely exposed, even down to the bulb. There is less chance of injury of the



facial nerve in this operation than when employing gouges and rongeur forceps.

7. Exposure of an extradural or brain abscess, can be accomplished either by continuation through the tegmentum mastoides, antri and attici, or by means of a removal of a

squamous bony flap, or an osteoperiosteal skin flap when not necessarily in the region of the ear or mastoid.

8. The typical Haynes operation can be performed for the drainage of the cysterna magna.

9. Decompression for tumors, abscesses, fractures, indentations, and intracranial hemorrhage can be performed.

10. Drill openings can be made for ventricular puncture.

11. The maxillary ridge can be removed in the submucous operation and the bony septum that is subsequently removed with forceps can be weakened. This avoids the possibility of fractures extending through the sphenoid into the skull or causing too much shock to the patient when breaking it off. The perfect safety to the mucoperichondrial flaps in this procedure is clearly demonstrated.

12. The intranasal operation on the antrum can be performed, the opening being made submucously in the inferior meatus, as suggested by Canfield, by means of the burr. The operation is much more rapid. A smoother opening is made for the adaptation of the mucous membrane flap.

13. In the Caldwell-Luc procedure with the writer's modification, the anterior wall of the superior maxilla is removed close to the margopyriformae without perforating the mucous membrane. This mucous membrane is incised close to the margopyriformae vertically and horizontally near the floor of the antrum, making an L-shaped triangular flap, which is held out of the way in making the opening into the nose. Both membranes, the antral as well as the nasal, in the region of the inferior meatus, are preserved to cover the raw bony margins of the opening that is made in the inferior meatus. This is accomplished by dissecting and turning temporarily the antral mucous membrane flap upwards. Sufficient bone is burred away to make the proper sized opening without perforating the nasal mucous membrane. This latter structure is cut close to the upper margin of the inferior turbinate and turned into the antrum, while the antral mucous membrane flap is turned into the nose under the inferior turbinate body. The flaps are held in place by stiff rubber tubing passing from the antrum into the nose. The L-shaped mucous membrane flap of the antrum is sutured to the submucous tissue of the gingiva margin at the primary incision. The opening in the anterior surface of superior maxilla shows below, the upper lip is closed immediately and the tube removed by way of the nose.

14. In the radical obliteration of the antrum, the same procedure is followed as described above, except that all the mucous membrane is removed from the entire antrum, except at the opening of the inferior meatus, which is treated as in the description given in No. 12. The bony surface of the cavity of the antrum is removed almost down to the soft structures, so as to have large granulations form for obliteration purposes. The cavity is packed with iodine and bismuth gauze and carried through the rubber tube in the nose and is repacked, until large granulations have formed, and subsequently bismuth paste is injected until the cavity is obliterated or nearly so. No opening is permitted to form into the mouth.

15. To reach the sphenoid and sella turcica by way of the antrum, Jansen's operation on the sphenoid, and the writer's advance to the sella turcica are followed. In this case a much larger portion of the anterior wall of the superior maxilla is removed in order to get an easier access and better view of the deeper structures. The ease with which the posterior ethmoid cells are taken down with the burr has led the writer to try it in intranasal work on the frontal, ethmoid, and sphenoid sinuses.

16. The external frontal sinus is explored, either for diagnosis or in acute frontal sinus disease to relieve tension, subsequently allowing this small opening to close.

17. The external radical frontal sinus operation is performed with the removal of all the mucous membrane, burring down close to the dura, enlarging the opening into nose, packing the cavity with iodine and bismuth gauze, which is passed into the nose through a stiff rubber tube and primary closure of the external incisions. Subsequently, after granulations are large, the cavity is filled with bismuth paste by way of the nose until the cavity is completely obliterated.

18. The superior maxilla is resected for malignant disease. This can be done by the burr without the mutilation of the face by any external incisions, by lifting the face as recommended by Loewe.

19. Kroenlein's operation or resection of the external wall of the orbit can be performed. This procedure is of particular value in exploring the retro-ocular or orbital region for tumor, abscess or foreign bodies. It is exceedingly easy of exploration by means of the burr with the least possible chance of injury to the vital structures. Especially is this true in suspected fractures through or near the optic foramen when the nerve is pressed upon.

20. Operation on the tear duct is performed by way of an external incision. An opening is made into the tear duct and then the nose at the inferior and inner surface of the tear duct at the level of the middle meatus. The drill opening is almost as large as the duct itself and is kept open by a fairly stiff rubber tubing, the face end of which is in the nose. This is allowed to remain for three or four days. The external incision is primarily closed.

21. Intranasal exenteration of the ethmoid can be done either by infracting the middle turbinated body towards the septum or removing the middle turbinate body first, before breaking down the ethmoid cells. With the light drill (Cryer) it is perfectly remarkable to see the ease with which the operation is performed. There is not any doubt that the lamina papyracea often may be taken away, and the periosteum exposed, but that does no more harm than exposing the dura in mastoid operation.

22. In the sphenoid sinus operation, after the middle turbinate body and ethmoid labyrinth have been removed, the anterior wall of the sphenoid can be removed with the light burr to a completeness and smoothness that can not be accomplished as well with any other instrument.

23. In the frontal-sinus operation by the intranasal method, follow the principles of Mosher in the entrance to the ethmoid by way of the agar nasi cell or that vicinity. It is as easy and safe to enter the frontal sinus with a light burr, slowly revolving, as by means of a curette or rasp. The precautions taken by Halle and Ingalls are not necessary, because the proximity to the inner table is less, besides injury to the dura is not likely, even though it may be exposed.

INSTRUMENTS USED

I have done most of my mastoid and skull work by means of the Victor motor and burrs. But, after experimenting on cadavers, I found that this instrument will not do for delicate and deep work as well as the Cryer machine. This latter instrument, which is practically universally employed by the dental profession, has the least vibration of any burr that I have ever used. In the heavy work, however, such as the skull and sclerosed mastoid, the Victor is probably the best instrument.

I have in the past year employed a new electrically driven instrument for opening the skull and making bone flaps which I believe is the most perfect machine yet made for that purpose. It is a chain saw, and one can make any shape or size

flap wanted without the slightest chance of injury to the dura. Drills and burrs may also be employed by the same instrument. It is known as the Carlson electric burr, trephine and saw.

DANGERS OF THE METHOD OF EMPLOYING THE ELECTRIC BURR

The much-talked-about danger of *burning the bone* is a myth from the observations that I have made by removal of pieces of bone and microscopic sections examined at different times after the experiment. The only place there is some danger of slight coagulation from the friction is in chronic suppurative otitis media in which a mastoid operation is performed. Here the bone is usually sclerosed and very little blood is contained within it. In such cases one should keep the bone moist by salt solution or allow the blood from the skin incision to flow over the part that is burred.

Perforation into the areas which subsequently may become infected, as the brain, sinus, eye, and neck is another danger. From personal experience on the living in operations and from experiments made on the cadaver, I find this danger is practically nil. What actually occurs is that the dura, sinus, periosteum, etc., are pushed away by the burr.

Both these dangers are possible, however, when the proper technic and instruments are not available.

PRACTICE FOR EXPERIENCE WITH BURRS

In order to learn how to steady the burr so that it does not slide or fly away from the parts that are being attacked, it is well to use a small bowling ball held in a vice. Cigar box wood is also fair material. The scapulae of animals make good bone to practice on and the great trochanter of the femur comes pretty close to the structural feeling of the mastoid. Dogs were the principal animals with which I experimented on the skull, but the bone is so thin in the greater portion, that it is of very little value. After all, by being careful one will acquire a perfect technic if only enough material is at hand in operations on the living.

DISCUSSION

DR. T. E. CARMODY, Denver: My early training was as a dentist, so I used the drill a great deal. The one I use at present is a belt-driven drill. Around the mastoid I have been deterred from using it on account of the objection of Black, of getting a facial paralysis, but have used it around the nose and mouth and find it very valuable, and think now I will try it on the mastoid.

HISTOPATHOLOGY OF THE FAUCIAL TONSIL

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DENVER

When we take up the bacteriology or the histology of the tonsil, we must first think of the work of Wood, Rosenow, Fetterholf, Wright and the recent work of Barnes.

The work done by others has been largely proof of the work of the first three named, and while I may add something new, it is the work which corroborates that already written which is most important.

It is not necessary that we go into the embryology or the relation of the lymphoid structures to each other, since these have been dealt with in a previous paper.

The method of removal of the great majority of these tonsils has been Sluder's method, as modified first by Beck (Fig. 1), who demonstrated the same to the author. The tonsil is delivered by this method without tearing or destruction and we believe gives much better sections.

Cultures from tonsillar crypts are of little more value than cultures from the surface, as the same bacteria are found in practically all cases. Streptococci, diplococci and staphylococci, although other forms are not infrequently found. There is, as has been stated, a possibility that many bacteria pass through the tonsil crypts to the lymphatic spaces and to the lymph nodes, without producing any pathological change in the tonsil itself, evidence of which we see very often. The changes in the tonsil, due to irritation, which is probably always bacterial, vary with the variety and virulence of the infection, the age of the patient and the portion of the tonsil infected.

Why should not every case of acute follicular tonsillitis be followed by peritonsillar abscess? First the plica may extend over the upper lobe of the tonsil to such an extent as to prevent infection. Should infection take place, the extension of the plica over the superior crypt or crypts becomes a hindrance to drainage and therefore an aid to infection of lymph spaces and nodes.

Although we most frequently hear the superior crypt spoken of, as though there were only one, we have found two or three crypts opening under the plica in a large number of cases.

Infection of the inferior lobe of the tonsil is not so likely to cause dire results, because we have good drainage and because in about 70 per cent. of the cases the capsule is not attached to the lower two-thirds of this lobe. Judging from this and from the fact that in the old cases in which we left the lower lobe very little if any trouble resulted, we may assume that most

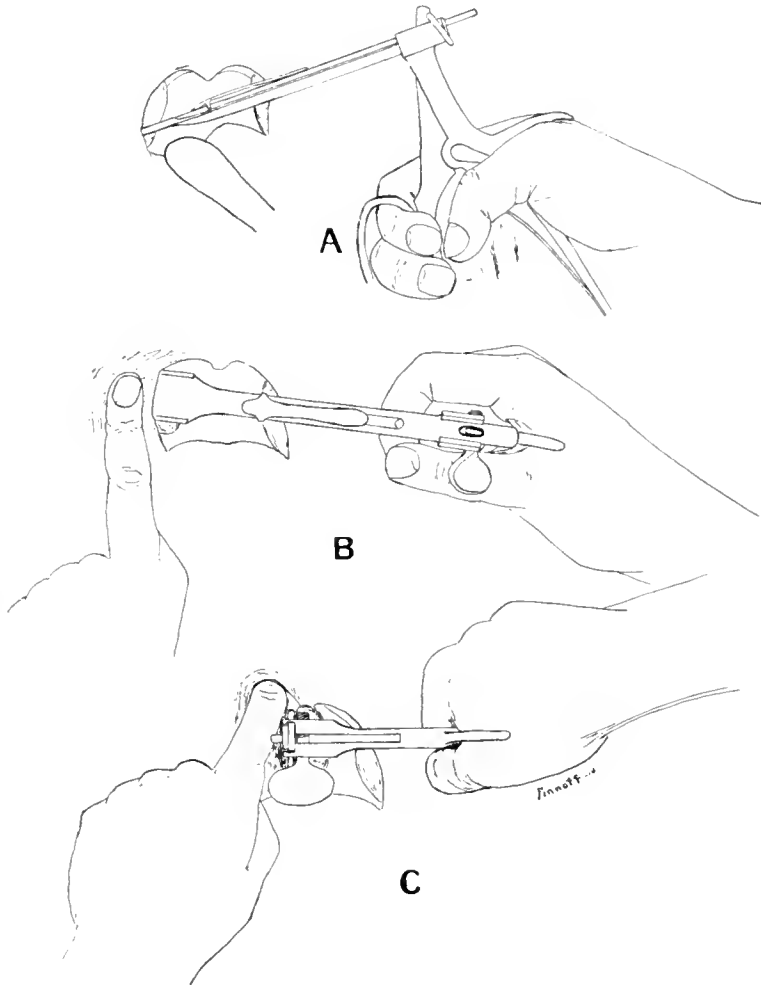


Fig. 1.—Beck's modification of Sluder's method of removing tonsils. Note position of the hand in (A) and instrument being placed so as to allow the lower lobe of the tonsil to pass through the fenestra while tongue is depressed. (B) Tongue depressor has been removed and instrument is rotated so as to face outward and forward while the index finger of the left hand presses against the oral surface of the anterior pillar, forcing the tonsil through the fenestra while the blade is pushed home. (C) Blade tightly closed and index finger of left hand is passed between the palate and the instrument then with a downward sweep all of the attachments of the tonsil to the fossa are broken through.



Fig. 2.—Muscle fibers in trabeculae.



Fig. 3.—Large blood-vessel between adenoid nests.

systemic infections which may be traced to the tonsil probably enter through the crypts of the superior lobe. Furthermore, the lower lobe is not bound down by pillars, except in the small percentage of cases in which the pillars are joined by an inferior plica, which encloses the tonsil in a sac, the mouth of which is incompletely closed, a state similar to which the upper lobe is usually found.

The burying of the tonsil is due, in my opinion, to the contraction of the connective tissue at the base, drawing the organ outward, and because connective tissue is stimulated by inflammation, the base of the tonsil is not only affected but also the lymphoid tissue and trabeculae, and later the pillars. As the tonsil is drawn outward, the increase in muscular fibers in the



Fig. 4.—Showing blood-vessels in capsule and trabeculae.

hypertrophied pillars contracts about its mesial surface and the increasing connective tissue in the pillars form a fibrous non-elastic ring which never allows the crypts to empty.

Many operators have noted that the hypertrophied tonsil is not larger than the buried tonsil. This fact has been proven by weighing the organs after removal, a method first resorted to by Coolidge and Garland. This only goes to prove that the hypertrophied tonsil is not a menace to any great extent, so far as infection is concerned, while the buried or submerged is a constant source of danger. However, there are other conditions which must be taken into consideration when speaking of



Fig. 5.—Abscess cavity in capsular region. Patient 8 years of age.

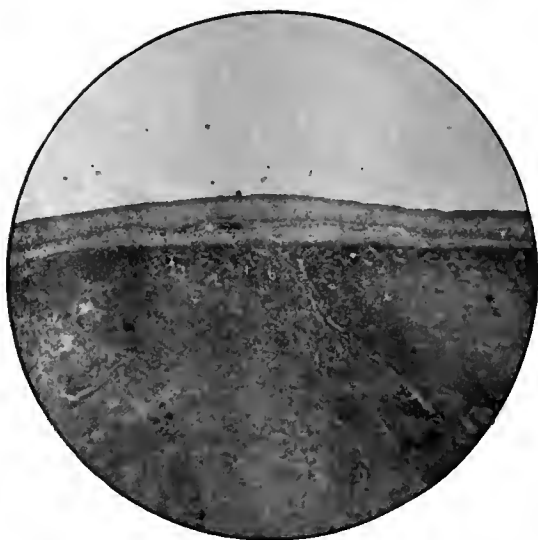


Fig. 6.—Basement membrane and epithelium covering internal surface of the tonsil.

infection as an epithelial barrier. This may be broken in many places allowing communication with the lymph spaces. It may and probably is very frequently broken by pressure of debris or by other mechanical means. The most likely cause of injury is through toxins devitalizing the individual cells, the pressure in such cases only driving the toxin deeper into the cell.

Whether infection can take place through intact epithelium is still a question. The epithelium covering the tonsil is seldom injured though we may find it entirely lacking just inside the mouth of the crypt and toward the distal end. We have also found a lack of basement membrane and a dipping down of epithelium into lymphoid structures.

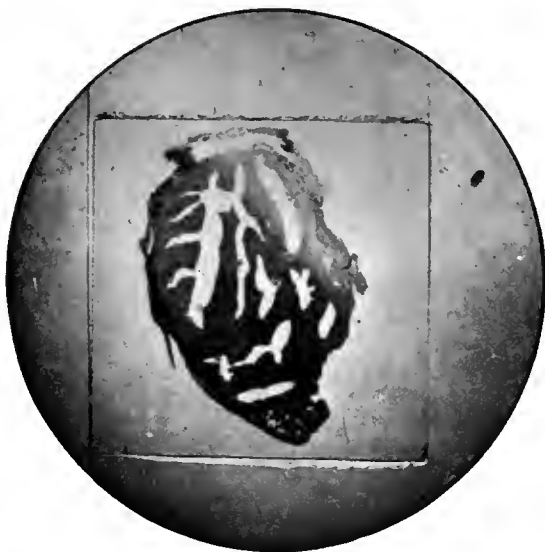


Fig. 7.—Macroscopic section showing dilated crypts and showing very few adenoid nests.

The pressure on the lymphoid tissue by connective tissue will necessarily cause an atrophy of the lymphoid structures, thereby lessening the combative power of the organ. The pressure is not limited to that made by connective tissue, but atrophy of lymphoid structure may be caused by epithelial plugs and debris in the crypts, as shown by sections, and yet we may find crypts widely dilated in sections with no atrophy.

The lymph spaces found near the crypts are connected with the small lymph channels of the epithelium and the brushing away of one epithelial cell gives you a direct channel to the lymph node. Many crypts are dilated, either by being filled with

necrotic tissue and bacteria or by a drawing away of one wall from the other by contraction taking place in the trabeculae. It is stated by Barnes that he never has found the walls in contact, the reason for this being that in most methods of fixing and imbedding tissues, slight shrinkage takes place and this necessarily enlarges existing spaces. We have found the walls in contact a few times, probably due to the fact the tissue is immediately placed in the fixing solution, but it is remarkable that only one or two crypts in the tonsil will be found in this way, others being sometimes markedly dilated. If we removed a perfectly normal tonsil we would find all the walls in contact



Fig. 8.—Macroscopic section showing a few dilated crypts and a number of adenoid nests.

excepting where the normal living lymphocytes intervene. There is a possibility that many infections of the cervical lymph nodes may be caused by primary infection of the nasal cavities, accessory sinuses, or from infection in the oral cavity. Infection of the tonsil may take place by infection following the retrograde stream after infection of lymph nodes as stated above.

It cannot be assumed as proven, that we have a direct lymphatic connection between the nasal cavity and the tonsil crypts in this way, neither can we prove more at present regarding the relation of the gums and tonsils.

We have, it is true, plenty of clinical proof and the experiments of Lenart, who found that after injecting insoluble color-

ing matter into the nasal mucosa of dogs, particles of the same were found in the tonsil as well as in the superior cervical lymph nodes and not only in the tonsil of the side on which the injection was made but in that of the opposite side. It remains, however, a task for someone to demonstrate the path along which these particles of infection pass; this can only be accomplished when some agent is found that will stain the endothelial lining of the lymph-vessels without penetrating the other tissues.

Many believe that voluntary muscle is not found in sections of tonsil unless part of the superior constrictor or part of the pillar happens to be included in the mass removed from the



Fig. 9.—Dilated crypts with no apparent atrophy of adenoid nests.

throat; but Figure 11 shows muscle bundles not only in the capsule but also extending into the trabeculae.

It is probably not necessary to mention that early in life we have pronounced activity in the lymphoid areas with marked proliferations of leukocytes and plasma cells. The activity in these centers gradually decreases as age advances and more connective-tissue cells, while not exactly taking their places, develop later into fibers which necessarily increase the space occupied by the trabeculae.

In inflammatory conditions we first have an increased activity in the adenoid tissue, with proliferation of cells. A little later an increase in blood-vessels in the trabeculae and capsule (Figs. 3 and 4).

The germinal areas then throw a large number of cells into the crypts. Leukocytes also gain entrance from the blood-vessels and these, with the epithelial cells already dead or dying and the wandering connective tissue cells, fill the spaces which contain more bacteria than they can devour or destroy; consequently we have either the formation of an abscess or slow and incomplete resolution with its resulting strangulation of vessels and adenoid nests.

Figure 5 shows a tonsil from boy of 8 years with abscess in the lower part of the superior lobe between the capsule and the tonsil proper.

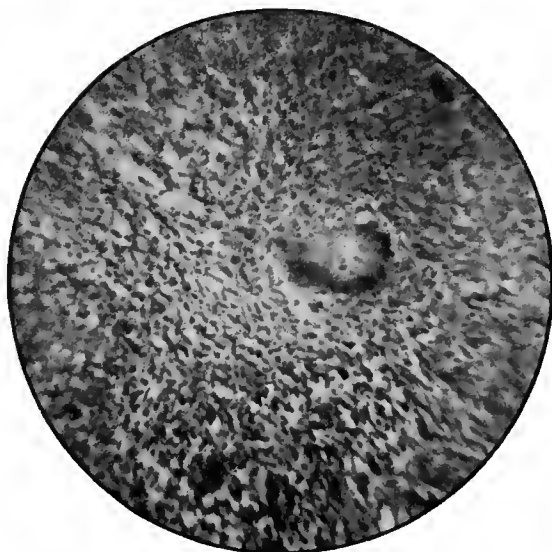


Fig. 10.—Giant cell in section from tonsil.

Figure 6 shows the basement membrane underlying the surface epithelium.

Figures 7, 8 and 9 show widely dilated crypts in the first; although the specimen is very thick, the lymphoid tissue has almost entirely disappeared; in the second only partially, while in the third practically not at all.

These are sections of the entire tonsils, and while the dilatation is partly due to plugs, it is largely due to the fact that they are celloidin sections, while those imbedded in paraffin are not dilated to such a marked degree.

While we not infrequently see tuberculosis of the tonsil, in the last stages of pulmonary tuberculosis, the primary form is rare, or possibly we should say not frequently proven. Figure 10,

from a girl 4 years of age, a patient of my associate, Dr. Lockard, had run temperature for weeks with enlarged cervical glands, following an attack of acute tonsillitis, the temperature disappearing on removal of the tonsils, and the glands gradually subsiding.

The following history is of especial interest:

C. K., aged 15, was examined by Dr. Hall, chief resident physician of the Denver City and County Hospital, on June 20. He was found to have large and diseased tonsils, with slight swelling on the left side of the neck, which had been present only a few days. He was told to return on June 24, to be operated on. During the interim he had fallen from the top of a barn, striking the left side of his head. On examination the tonsils appeared about as before, but swelling of the neck had increased. The patient was kept under observation for one week, when, there being no change, tonsils were removed under chloroform anesthesia. No improvement took place in the neck and three weeks later I made an incision from the tip of the mastoid to the clavicle and removed the glands, the whole chain being

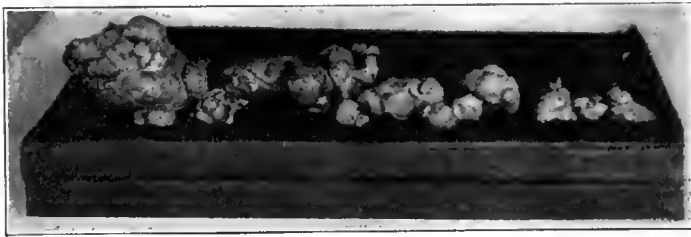


Fig. 11.—Glands removed from boy of 15, which were proven on microscopic examination to be tuberculous. Probably infected through tonsil.

involved and adherent to the carotid sheath. Examination proved them to be tuberculous and markedly caseous. This was apparently an acute tuberculous process, which would probably have run a chronic course except for injury.

In this form of tuberculosis of the tonsil we probably have a type which frequently heals, or one that at least takes years to develop. I have never seen or known a case where ulceration has taken place, although giant cells are frequently found. A large number were present in the slide from which Figure 10 was taken, although no caseation was found. Neither can we demonstrate tubercle bacilli. Barnes fully agrees with the latter statement.

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DISCUSSION

DR. CARMODY (closing discussion): I only wish to thank Dr. Beck and Dr. Kopetzky for discussing the paper. Since arriving here I received a letter from my assistant saying he had examined a case which we had operated only about a week before I left home and found a giant cell. He forwarded a photograph of the same, which I am passing around. As to percentage, this is always a little high it seemed to me, and so this examination has been very thorough. We have taken out every tenth, twentieth or fiftieth section and examined it, and if we found anything remarkable, we made serial examinations. In this one case Dr. Finnoff said he had examined it further, but could find no further giant cell. This microphotograph shows a very large number. We have examined very carefully between 125 and 150 tonsils and have gone through them very thoroughly, not always both tonsils, but if there was any question we took the other tonsil and sectioned it as well.

VACCINE THERAPY IN EAR DISEASE

A FURTHER CONTRIBUTION TO THE STUDY OF THE SUBJECT

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WASHINGTON, D. C.

Stengel comments on the slow acceptance of diphtheria anti-toxin by the profession at large despite the exhaustive study and scientific clinical demonstration, as contrasted with the eagerness and indiscriminating favor with which all sorts of vaccines and serums are now employed. This haphazard zeal has not been restrained by the natural check of ill effects, as there are practically never any, though Stengel is not willing to admit this. I have never seen a case where the use of vaccines could have been instrumental in any untoward result existing after their use. The unthinking use of mixed vaccines and stock preparations generally reflects no credit on the attendant, usually results in no benefit to the patient and frequently does harm to a legitimate therapy, which has a field of value, restricted though it be.

A suppurating ear with necrotic bone as a base is manifestly an improper field for this treatment, though it is so employed frequently, either through ignorance of the merest fundamentals of pathology or lack of a proper interest in the study of the case. The inevitable failure should bring discredit upon the otologist, not upon the therapy. On the other hand, the opsonic index, with its complicated technic and the dreaded negative phase, deterred many from using vaccines, whereas we now know that it is safe to ignore them both, watching vigilantly instead the clinical signs and symptoms as a sufficient guide. In this opinion I find myself in accord with Holden, who thinks that over the whole field of medicine the failures are more frequent than successes. In ear disease this is not true, as thoughtful otologists do not use vaccines in every suppurative otitic disorder, nor as the only therapeutic measure. Vaccine therapy is distinctly and only an additional measure, and never to be undertaken to the exclusion of any and all the usual precautions. Moreover, even with this reservation, success depends much on the culturing, preparation of the vaccine and its method of administration; thus creating a wide margin of error, and by the same token, of failure to benefit. While I do not wish to enter on the bacteriologic technic, I will add the final touch by

saying that a vaccine is a delicate substance and one easily destroyed by a few degrees of heat in excess of that required to secure sterilization. A laudable zeal to attain this essential at times leads, I am sure, to a sterile vaccine, it is true, but also to an inert preparation. Thus again we see another cause of failure.

In chronic suppurative processes of the ear, it is not always easy to secure the causative organism, largely due to reinfection on top of the basic trouble and the attachment of saprophytes to the decayed material. For this reason repeated reculturing both before and during treatment is necessary if we are to be sure of our bacteriologic diagnosis. Acute suppurative otitis media generally clears up so readily under the ordinary cleansing treatment that the temptation is strong to omit vaccines from the therapeutic armamentarium, yet the reports of McDonald, Weston and Kolmer, Conners, Christie, Jacobs and Dabney show that their use was of assistance in the cure of three times as many acute cases in thirty days as was experienced without this aid. Moreover, they were the product of the more virulent infections, such as scarlatina, influenza, typhoid fever, and diphtheria. Three years ago I used vaccines extensively in acute otorrhea, but I have since then modified my use to this extent, that I omit children of a neurotic strain, and wait for five to seven days before starting the treatment. This latter precaution allows the discharge to give some intimation of its virulency, and consequently, necessity for vaccine. Moreover, it is known that a vaccine acts better after this pause than if given earlier, before nature has well organized its phagocytic defense. Thus, it sometimes becomes apparent that the disease will yield without the use of vaccine.

Where it is at all possible to get it, the autogenous preparation should always be employed; this has been firmly established, not only as a scientific measure, but on grounds of efficiency (Wolfsohn, Kreuscher, Wright, Pearce, Dabney and many others). The use of a stock preparation (save in one apparent exception to be noted later in my own work) is a leap in the dark and unscientific in the extreme.

The following are the organisms from which I have vaccines prepared, and experience has demonstrated to me that it is useless to experiment with other organisms, a lesson I have learned only after much onerous research. The doses are based on the adult, to be reduced proportionately for the young: *Staphylococcus pyogenes*, *aureus*, and *albus*, 250 million; *Strep-*

Staphylococcus pyogenes, 25 million; *Bacillus of proteus* type, 30 million; *Bacillus pseudodiphtheriae*, 40 million. Injections are to be repeated every three or four days and increased one-third, depending on the reaction and the progress of the disease. Nephritis, diabetes, tuberculosis and severe constitutional depletion are contra-indications. It is always wise to give an additional dose after apparent recovery, as it tends to prevent a relapse. Failure on the part of the patient to submit to this precautionary measure has caused just this setback in my practice several times.

TABLE SHOWING RESULTS OF USE OF AUTOGENOUS VACCINE IN CASES OF SUBACUTE AND CHRONIC PURULENT OTITIS MEDIA AND MASTOIDITIS

Author	Disease	Number	Cured	Improved	No Change
McDonald	subac. O. M. P.	13	13
	chr. O. M. P.	17	3	5	9
Weston & Kolmer	subac. O. M. P.	100	83	10	7
Patterson et al	subac. O. M. P.	10	7	0	3
	chr. O. M. P.	17	3	3	11
Connors	subac. O. M. P.	7	2	3	2
Christie	subac. O. M. P.	6	6
Jacobs	chr. O. M. P.	6	2	4	..
Thomas	chr. O. M. P.	1	0	0	1
Hoobler	chr. O. M. P.	2	2
	mastoiditis	1	1
Beck	chr. O. M. P.	7	0	7	..
Scott	mastoiditis	1	1
Sill	subac. O. M. P.	124	70	22	5*
Dabney	Furunculosis	36	36
	chr. O. M. P.	22	7	5	10†
	subac. O. M. P.	23	18	0	5
	mastoid sinus	15	9	4	2†
* SUMMARY					
	subac. O. M. P.	260	181	35	17
	chr. O. M. P.	69	15	24	30
	Mastoiditis	1	1
	Furunculosis	36	36
	Mastoid sinus	15	9	4	2

* 3 operated for mastoiditis; 23 disappeared.

† Two diabetics.

In a paper some three years ago I invited attention to one of the incidental effects of the use of vaccines, namely, the metabolic improvement and bettered physique. Similarly Bruce noted in patients afflicted with chronic mania, in whom lack of nutrition so often is a prominent symptom, that the use of autogenous vaccines was nearly always followed by increase in weight and improved metabolism. This same corrective influence is often observed under the administration of vaccines in the course of a stubborn middle-ear discharge, but is not to be accounted for solely by the cessation of the suppurative process (as it does not always cease), since metabolic changes are ordinarily slow after the local lesion ceases to poison. In long suppuration of the middle ear, and in stubborn sinuses or unhealed wounds following the various mastoid operations, there is encountered often a septic condition, almost a cachexia, in a

patient whose anemia, depleted vitality and loss of appetite are familiar to all otologists. The correction of this unnatural state and usually of the pus formation too is with the assistance of vaccine the result of one and the same process, which I interpret as an elaboration of the side-chain theory of Ehrlich. In brief, the receptors previously having in health the function of uniting with a food molecule and assimilating it, under disease conditions unite with a molecule of toxin, thus shutting off that much nutrition from the body. Naturally when the toxins are numerous, numerous receptors are preempted by them, and the metabolism of the body suffers proportionate detriment. Thus, in destroying these toxins, the vaccine renders available for food assimilation receptors heretofore occupied with toxin assimilation.

Furunculosis of the auditory canal is an unusually stubborn, painful affection, lasting frequently from ten days to four weeks, but I have seen only one case which lasted as long as a week under vaccine treatment, and required as many as three injections (whereas I have seen a case of two months' duration cured after two injections). With this exception, all my cases have cleared up in less than six days and with only two injections; consequently I regard vaccine as a specific for furunculosis of the canal, save where it is caused by diabetes, lues or tuberculosis. The first injection generally relieves the pain in twelve hours, and the swelling is markedly less; the second dries up the discharge, if there is any, and causes all tumefaction to disappear. This result cannot be explained by the increased drainage in those cases which are incised, as incision alone has not so benefited other cases; moreover, some of them had already ruptured, others had not, and did not rupture. Again, we see cases which are flowing freely where all the symptoms continue despite the good drainage. Such confidence have I in the treatment here advocated that I have been able twice to diagnose diabetes from failure on the part of vaccine to help cases of furunculosis, though the internist who referred the case had no reason to suspect the glycosuria, nor had I till the injections failed to benefit. The following case should convince the most doubting of Thomases:

CASE 1.—J. D., a boy 4 years of age, had been suffering for several days with earache and swollen face; when seen June 11, right canal was so tightly closed as to admit probe only with the use of considerable force; whole right face was swollen and red and brawny; lower eyelid edematous and eye nearly closed; temperature 100 F. Canal incised, half dram of pus escaped; 100

millions stock staphylococcus vaccine injected. Next day, June 12, a little more pus squeezed out; eyelid and eye practically normal; brawny area gone save pretragal gland; no pain; canal patulous, permitting view of membrana tympani, which was normal; felt so well that he did not return for six days, contrary to my orders; drop of pus wiped out of sinus; 150 millions stock vaccine injected. Had he received this three days earlier according to routine there would have been no pus and no sinus left. June 22 he was still well; no relapse. Case healed and well in all respects June 20.

The culture showed pure *Staphylococcus aureus*, and stock vaccine was given, as it takes from four to five days for the culture and the preparation, and the cases all yield as a rule before this time. However, the stock is not the ordinary commercial article by any means, but is prepared in the laboratory of the United States Army Medical School, and is drawn from seventeen strains of unusual virulence. This is only an apparent exception to the rigid rule to use autogenous vaccine, and to be classed as one of the times when it was impossible to secure it.

Instead of giving other clinical argument, I will say that I have seen a case of six weeks' standing clear up in a week with two injections, and without any other treatment whatsoever, but with the before-mentioned improvement in physique and color.

Where there is caries of any part of the auditory tract, vaccines can effect no permanent good; where the discharge originates from the soft parts, their use can be of service, if the causative organism is isolated and secured. Herein is briefly stated the province and limitation of vaccine therapy in chronic suppurative otitis media, and in sinuses following operation on the mastoid (Dabney). The granulating surfaces left in the latter class of cases seem prone to become sluggish and at times the cause seems to lie in reinfection or in a lowered resistance to that infection which has always existed.

CASE 2.—M. E. H., a child of 6 years, had been operated on for acute mastoiditis and four months later the wound had been curetted for failure to close. Seven weeks after this curettement she was brought to me, when the wound was observed to be 2 inches deep, $\frac{3}{4}$ of an inch in diameter and cone shaped. Of an autogenous vaccine, prepared from the culture of bacillus pseudodiphtheriae, 10 millions were given. After two injections, the granulations which had been pale, flat and glazed, not bleeding on manipulation, became raised into papillae, bright red and easily made to bleed at the slightest touch. After an interval of six days an injection of 30 millions was made, and the wound was then found to be $\frac{1}{2}$ inch less deep; after another pause of ten days 40 millions were given. Here we had to suspend vaccines as the child had become highly intolerant of the pain caused

by the needle. However, the impetus given the healing process was sufficient to close in five weeks a wound that had not healed in five months, and had shown no evidences of the reparative process for two months before the use of vaccines.

CASE 3.—E. D. M., woman of 23 years; radical mastoid done four and one-half years ago, never dry, though narrowed to a two-limbed sinus for past two and a half years; facial neuralgia practically continuous for three years; pale, sallow, neurotic, verging on melancholia, 10 pounds underweight. On removing the cholesteatomatous deposit, a pure culture of *Staphylococcus albus* was obtained, and autogenous vaccine was given at intervals of a week and later two and three weeks, the dose beginning at 250 millions and carried up to 800 millions. Treatment covered a time from Oct. 15, 1911, to Dec. 22, 1911, with complete cessation of the discharge and firm healing, though the scar broke down once May 4, 1913, and July 1, 1914, only to heal, under cleansing with peroxid and argyrol, in a week. The neuralgia disappeared in the first four weeks, returned twice a year later, but on drawing two carious wisdom teeth permanently disappeared. Patient became a totally different person, cheerful, gained ten pounds in weight, has had no trouble save an occasional collection of hard wax mixed with detritus which requires only one treatment for its relief. The only internal treatment given was phosphatic emulsion of cod liver oil, which I employ in all mastoid cases which show any tendency to prolonged convalescence or caries.

Labouré reports a similar case, in which the vaccine assisted the convalescence after a radical operation for the cure of an otorrhea of twenty years' standing. The operation alone did not accomplish the desired end until vaccines were employed.

CASE 4.—H. H., woman, aged 24, good general condition; bilateral suppurative otitis media for two months; similar attacks every winter for several years; never such severe pain or profuse discharge as now. Very deaf, and mastoid quite sensitive on pressure over antrum; long polyp removed from left canal, which it completely filled, attached to drum. Two hundred and fifty millions autogenous *Staphylococcus aureus* given; discharge ceased in eight days; second precautionary dose on eighth day; no return; deafness relieved by six inflations of tube.

CASE 5.—Exhibits the result of omitting the precautionary dose. V. T. T., woman, 32 years of age, acute running ear for two weeks; 250 millions autogenous *Staphylococcus albus* vaccine given and discharge ceased in ten days. Two weeks later discharge returned, as patient received but one dose; a second dose of 350 millions resulted in a cure in four days. The deafness, almost absolute in the affected ear was relieved by the routine inflation.

Some of the cases given herein to strengthen my contentions have been reported in another paper, and some of the text is

of necessity a quotation from it. The accompanying table needs no explanation, but is given as the most concise manner of summing up the results, which, after all, are like wisdom, "the principal thing."

While I have given somewhat in detail only the successful cases, as the unsuccessful ones require only a record of failure, the table will show signal failures in abundance. In commending this therapy, let me repeat that I have no illusions on the subject, and that it is no royal road to health for afflicted ears, nor should it be used without all the other means of treatment commonly employed by the experienced. Every case I have ever handled has been given the most searching routine treatment known to us all as of benefit, but I have not so stated it in each case, as it was deemed unnecessary.

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VOICE FATIGUE IN SINGERS AND SPEAKERS

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NEW YORK

This subject has been so fully and intimately explored by German authors, notably Flatau, Imhofer and Gutzmann that I can scarcely hope to add much from my own relatively meager experience to our stock of common knowledge. However, since there seems to be a dearth of information concerning voice fatigue in singers and speakers in our American periodical literature, I have thought it not unwise to present some of the facts which I have been able to gather from a rather thorough review of the literature and from an extensive study of such cases as have come to my attention.

Voice fatigue was recognized as an entity as far back as 1600, when Fabricius ab Aquapendente expressed his views on the subject in a rather laborious essay. In England the condition was long known as clergyman's sore throat, while in France it was named more accurately, called *fatigue de la voix*. Not until 1906, however, was it scientifically and accurately described in a little brochure entitled, "*Die funktionelle Stimmeschwäche (Phonasthenie) der Sänger, Sprecher und Kommandorufen.*" In that monograph Flatau not only gave a clear exposition of the entire matter but recommended and described a rational and successful therapy for the same.

Weakness of the voice, or phonasthenia, as it is now commonly known, is a disturbance in which a given voluntary impulse to the vocal bands is not followed by a normal tonal effect; that is to say, the produced tone is higher or lower than the intended tone, is unpleasant to the ear and has no staying or carrying power.

The fundamental cause of this difficulty is in many cases faulty voice placement. Just as many people never learn to walk gracefully, so they also never learn how to get the most out of their voices. In America we seem to be particularly unfortunate in our speech habits, so much so, that our so-called nasal twang and careless pronunciation make us a butt for ridicule in many foreign countries, where we go to exchange good American dollars for as much Old World culture as we can assimilate within a few short weeks.

As an expressionist friend¹ has very tersely put it: "Americans returning from Europe hear for the first time the raucous, sharp, high-pitched, nasal, unmusical voices of their countrymen. Their national self-complacency has been punctured, and they set about correcting what to them has become an offense.

"The actor has always recognized the need for a pleasing expressive voice under perfect control. Ministers who deal with the greatest subject that has ever engaged the mind of man—the destiny of the human soul—pay practically no attention to the method of delivery of their message. They rise on their toes, fold their fingers, and scream till they are purple in the vain effort to be impressive."

To obtain the best effect, one must study the art of control over body and voice. This demands laborious and painstaking effort. The time to begin is in childhood, at the "awkward age," when thought processes are still plastic and have not reached the stubborn and sometimes almost hopeless automatic stage. The inadequate term *elocution* has in recent years given way to "expression" or "interpretation," and much is to be hoped for in furthering the cause of proper vocal technic.

Voice is produced not by the throat as is commonly inferred, but by every part of the body. The nose, accessory sinuses or resonators, pharynx, buccal cavity, teeth, lips, tongue, larynx, lungs, bony thorax, diaphragm, respiratory muscles and most of all, the central nervous system take part in this complex vocal mechanism. The ears give one an idea of quality, timbre, pitch, loudness, etc. Touch enables one to "feel a tone," as the expression is. The muscular sense is capable of telling whether individual muscle groups and antagonists are working properly.

As for the throat itself, correct function of the vocal cords calls for the purest and best tone consequent on the smallest output of effort consistent with artistic speaking and singing. This is a fundamental law and the one which is most frequently violated. The campaign speaker is always confounding big, burly voice with strong argument, and the ambitious singer is always mistaking a big, brawling tone for genuine artistry. Accuracy of method should be the first consideration.

Phonasthenia is a condition which affects nearly all ages and both sexes. Voices of high pitch are especially prone to fatigue, because they not infrequently have poor carrying power, and

1. Miss Theodora Ursula Irvine, of Carnegie Hall, New York.

the user is always making an increased effort to be heard distinctly by all.

Teachers, preachers, stump speakers, vendors, telephone operators and singers are most frequently affected.

Certain physical causes associated with disease conditions engender disturbances which prevent the muscles concerned with voice production from reacting properly to normal impulses. Thus anemia and chlorosis, through insufficient nourishment of the muscles and improper removal of waste, are not uncommon causes.

In such cases, where voice gymnastics and other therapy fail, arsenic, iron and strychnin may work a cure.

The edges of the vocal cords are extremely sensitive to slight influences. For example, pregnancy, a physiological condition, may change the quality and carrying power of the voice very markedly. Menstruation also affects some women in like manner.

Convalescents from typhoid and influenza frequently show marked vocal changes which are for the most part, however, merely temporary.

Chronic diseases are a fertile cause of voice fatigue. In this connection chronic tonsillitis with concrement formation is especially important. Nasal growths and deformities, purulent discharges, and chronic hypersecretion are also frequently responsible agencies.

Besides the above-mentioned, we have also to reckon with etiologic factors which demand a greater amount of strength for the production of a tone than should be employed. False teaching and improper vocal efforts are common factors. Imhofer says that two-thirds of all cases of phonasthenia in singers is due to bad schooling. Flatau believes that 80 per cent. of all young singers are affected with phonasthenia by the end of their third year of study.

There seems to be no one error of method, but every rule which violates the law demanding the use of the smallest amount of effort for the maximum of effect tends to phonasthenia.

The symptoms of phonasthenia are definite and certain. There is sudden and severe hoarseness, huskiness, tendency to clear the throat constantly, pain in the sides of the neck, and pain on swallowing. There is no sign of an active inflammatory process, although the redness due to trauma is pronounced if the condition comes on acutely, as in public speakers. The voice

is weak, unsteady, tends to break in certain spots and slide off the pitch into a lower key.

If the patient is a singer, one should go carefully into the history. Usually there have been from two to twenty different singing teachers, each of whom has told the pupil that his method was all wrong, and must be utterly changed. This produces so much confusion and uncertainty that the patient will usually confess to a lack of knowledge of the very fundamentals of voice production.

Many of these patients have gone through a process of having the voice screwed up (*hinaufschrauben der Stimme*). A baritone has been changed into a tenor, or a contralto into a soprano. In some borderline cases it is almost impossible to say which type one is dealing with, whether, for instance, a voice should be trained as a baritone or a tenor. Moreover, certain peculiar changes take place quite unexpectedly at times. Thus Caruso began his career as a baritone, but his voice changed naturally to an exceedingly remarkable tenor. In any case, singing out of one's range has ruined many a good voice. Some music, as written, is especially difficult even for a well-schooled voice. For example, the character, Ortrud in *Lohengrin*, makes very severe demands on an alto.

Breathing, while essentially simple, is a great bugaboo to many singers. Instead of being an involuntary automatic act, it is forced voluntary. The fault is chiefly in improper expiration. A singer will fill the lungs with air when about to take a high note and strive to force the entire volume through the narrow chink of the glottis. The Germans aptly term this, singing *mit wilder Luft*. The column of air is too rapidly discharged. Here the abdominal respiratory curve is too sharp. It should be wide and show a gradual approach toward a straight-line effect.

The much discussed *coup de glotte*, stroke of the glottis, is considered by Imhofer to be a primal cause of phonasthenia. One must distinguish between this method of attack and that used by coloraturas in doing agility exercises; for the latter is physiologic, but the former demands that the vocal cords be pressed tightly together, a condition dependent on faulty breathing, as we have just seen.

Tongue interference is also a common cause of voice fatigue. Here the tongue is arched upward and backward, pressing against the epiglottis and dampening the desired effect to a marked degree. Usually, the more the teacher draws the pupil's

attention to this matter the more unruly does the tongue become. Holding the tongue down with a pencil or spatula does no good. The condition is of psychic origin and must be corrected from that viewpoint.

Nearly every teacher has some special trick or device for overcoming all the singer's difficulties, which he has elaborated into a "method" and promulgated as a law. There is only one way to sing and that is the correct way, viz., by using a minimum amount of effort to produce a maximum artistic vocal effect. Singing is a great art, which does not readily yield to a series of rules and regulations. Because it is such a simple, natural process, excessive thinking about how it is done has made it one of the most difficult arts to acquire. It is first of all a gift—a gift which is not widely distributed among mankind, in spite of the fact that nearly every one can sing a little. A high artistic standard has been set by the world's great singers, which is impossible of attainment for the great mass of mankind. It is a gift which manifests itself in infancy, and hard work is only a very subordinate qualification in attaining great place in the world of song.

For years the battle has been waged between the so-called open technic of the Italian school and the closed technic, which is more distinctly French. This debate is of some importance to us as physicians, because it has a direct bearing on phonasthenia. A combination of the two would be ideal.

Singing with wide-open mouth, enormous lung volume and violent movements of the body lays the foundation for an early onset of phonasthenia. It is especially bad for alto, baritone and bass voices, where "big tone" is considered of primal importance. On the other hand, the excessively "covered" type of production, projecting lips, cramped smile and a too frequent use of pianissimo wearies the listener and robs the voice of that dash and brilliancy so essential in certain types of song.

Anything which disturbs the automatic singing act, every adventitious element in the tone-producing and tone-resonating apparatus violates the fundamental principle of the least exertion for the securing of the greatest effect and tends to the production of phonasthenia. The voice must be handled as an individual problem. The psychic element, mental poise, and suggestion are all-important. It is very necessary for every one who essays to deal with singers to cultivate an attentive ear, refined taste and a habit of reflection. Only in this way can

critical judgment be educated to interpret what is wrong with the voice.

Inhofer has put the matter tersely and correctly: "*Wer sich also mit Singstimmenbehandlung abgeben will, muss Laryngologe, Musiker und Diplomat sein.*" Whoever busies himself with the treatment of voice defects must be a laryngologist, a musician and a diplomat all in one.

The treatment of voice fatigue both in speakers and singers is fraught with difficulty, chiefly because the thing most to be desired, viz., rest, is felt to be impossible. The patient wishes to know how he can go on with his work and make a fair showing in spite of his disability. But it is of no use to treat such a case and let the causative agent go on. The physician must recognize all causes for the vocal breakdown both direct and contributory, and deal with them accordingly.

With singers, a system of prophylaxis should be worked out. This will surprise most persons, for such a thing as hygiene of the voice is practically unknown. The teacher must be invited to send his pupils for an early diagnosis, not only of the nose and throat, but of the general condition as well. This should be done at the first interview between teacher and pupil, before lessons are begun. If in the general examination there are extreme nervousness and irritability, poorly nourished body, chlorosis, anemia or debilitating disease, the pupil should be advised not to undertake so difficult and prolonged a task as voice culture. If he does, disappointment and even worse is inevitable.

The "feeling" and significance of early voice fatigue should be explained. Lessons should last not longer than twenty minutes, consisting of vocalization for five minutes followed by a rest for five minutes. The pupil should not go home at the end of the first lesson and strain his voice by attempting arias from grand opera. He must be taught how delicate the vocal mechanism is, and that a long, bright career is rather to be desired than a short, brilliant one.

On his own behalf the physician should realize the utter futility of spraying, painting, burning, use of pastilles, etc., save when called for in acute infections and chronic productive processes.

When treatment is decided on, the full and earnest cooperation of the patient must be insisted on. Discipline must be rigid, even to the minute of the hour for appointment. First of all a voice analysis with piano should be carried out and notations

of all defects made. Then the "silence treatment" must be carried out rigorously, remembering, however, that this will not cure voice fatigue, for the symptoms will disappear only to return when the voice is used.

The patient is better off away from people who would make social demands on him. In acute cases vocal gymnastics can be begun right away, but the general body condition must be good, especially the nervous system.

Local causes, growths, etc., should be removed only when a functional analysis shows that they are of great importance. One must remember that these patients are usually suffering from local over-treatment. A specific case of this came to my attention, in which a phonasthenia had been so much operated and cauterized that he became neurasthenic as well, so that he could not even take vocal gymnastics. Nasal insufficiency, if marked, leads to forcing and should be corrected, but preferably by a conservative method.

Large tonsils in singers must be studied before removal is advised. It may be wisest to do a tonsillotomy rather than a tonsillectomy, or even to treat the crypts conservatively.

Caustic agencies are to be avoided in phonasthenia. Silver nitrate should not be used in the larynx unless there is a chronic laryngitis. It does no good in phonasthenia and is very disagreeable to the patient. Warm applications are useful for pains in the throat. Menthol may be applied to the mucous membrane for cooling analgesic effect.

In 1908 Flatau advised cupping each side of the larynx for from fifteen to forty-five minutes. After a few hours this is followed by a moderate edema and swelling of the vocal cords. In a few days the cords become paler, less swollen and of greater mobility. This method often does well with speakers. A spray of ice water is a welcome measure with some persons.

Voice fatigue in speakers is often due to the fact that the voice is pitched too high; i. e., above its normal range. According to Spiess, the most favorable tone register for speakers is about three tones below the middle of the voice range. During an attack, stasis by external cupping or a constricting neck band is of value. Meyjes advised a spray of ichthyol, 5 to 10 per cent. As soon as possible the patient should be taught by a teacher of expression how to secure and maintain a proper relationship between the natural voice and the height necessary to declamatory demands.

The speech must be slow, fairly light, with good lip and tongue action. The voice should be directed forward against the upper teeth and hard palate, and increased and diminished in a monotone. Certain syllabic exercises, such as the no, na, nu, ni, na and co, ra, mo varieties, sung with moderate strength in middle voice, are helpful.² During these exercises especial attention must be paid to the breathing. It is of course impossible for the busy physician to undertake this work personally, but he should insist that it be carried out with a good teacher.

Inhofer and others have worked out a helpful, but rather complicated, system of electrical tests of phonasthenia, which need not be taken up here. Such tests are of value chiefly in the resistant types of the affection and in long-standing cases.

When can one safely say that he has cured a phonasthenia? This is sometimes a difficult matter to determine, but if there are no subjective disturbances, if the voice is of good, clear quality and keeps to the pitch—in short if there are no disturbances of intonation, we may safely dismiss the case. A useful test is made by applying an electrical current to the larynx during vocalization on a single note. If the larynx is normal, the tone should be elevated because of increased muscular action.

Quite marked phonasthenic symptoms may disappear under proper attack, correct breathing and accurate tone placing. Simple exercises have sufficed to reduce vocal nodules and general cord thickening when these are not too marked and of too long standing.

In conclusion let me say that the entire voice problem is worthy of intensive study at the hands of every laryngologist. Our advice and help is badly needed by the singing-teacher, and we in our turn can learn very much of interest and practical value from him. Cooperation should be our watchword.

DISCUSSION

DR. OTTO GLOGAU, New York: The question of phonasthenia, although rather new in this country, has been in Europe a matter of study for several centuries. The first scientific article was written by Fabricus ab Aquapendente in the year 1600. He dealt very thoroughly with the so-called clergyman's sore throat. The most important discovery was made by Michaels in 1876. He found that pressure on the cricoid cartilage raises the pitch of the middle and falsetto voice, while it is again lowered when the pressure is released. He also found that pressure on the pomum adami lowers the pitch of the middle and chest voice. This discovery

2. Credit for these exercises should be given to the well-known American vocal teacher, Mr. Edmund J. Meyer.

is to-day the foundation of the treatment of phonasthenia. Since Flatau, we use the electric current to diagnose and treat this functional disturbance of the voice. Dr. Skillern mentioned that in many cases adrenalin restores the voice. These are not real cases of phonasthenia; they are probably acute laryngeal catarrh.

Voice culture has been emphasized in the school. The children are forced to sing choir songs; that may be suitable for some children, while in a great number of them the range of the voice may not be sufficient. This abuse of the singing voice is the first step toward the development of phonasthenia.

The essayist is to be congratulated upon bringing this important subject before this meeting, as it is one every laryngologist ought to be familiar with.

THE CONTROL OF HEMORRHAGE IN TONSILLECTOMY

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It would seem from the scarcity of literature on tonsil hemorrhage that its consideration by the profession is either so unimportant as to deserve no mention, or its control so easy as to need no description.

However unimportant tonsil bleeding may have seemed to the profession generally, in my opinion it is one of the most important and most dreaded features connected with the surgery, not alone of the throat, but of any part of the body. The fatalities that have occurred from this cause—and they have been much more numerous than would appear from a survey of the medical literature—have received wide-spread newspaper notoriety and have remained vividly impressed on the minds of the laity as a terrible nightmare that may make its appearance in any tonsillectomy. To the physician himself, serious bleeding is, I believe, of greater importance than he has cared to admit.

This is the sort of emergency that calls for immediate attention and constant attendance until the bleeding has entirely stopped, and if the loss of blood has been profuse, until the immediate effects of this have been counteracted. The extreme exhaustion, the profound anemia, and the aspiration of blood pave the way for pneumonia as well as for every sort of infection in the nose and throat and their accessory sinuses.

The frequency of serious bleeding is variously stated by different men. This apparent discrepancy is explained by the several possible interpretations of the word serious. In my own experience of about 3,500 cases, serious hemorrhage has occurred on an average once in every 250 cases, much more frequently in the early cases and much less often in the more recent ones.

Hemorrhage that is troublesome and may become serious if not recognized on the table occurs very often, probably once in every three or five cases. This is the sort of bleeding that is readily controlled either by digital compression or by the application of ordinary artery clamps, as described below.

By serious, I mean bleeding that has been sufficiently profuse either to warrant the suturing of the pillars at the time

of operation or to necessitate the return of the patient to the operating room for this purpose.

The term primary hemorrhage should be restricted to such cases as result from a continuation of the bleeding necessarily incident to the operation; whereas the secondary type is a recurrence of bleeding coming hours or even days after the throat has been free from blood and caused by either the dislodgement of a clot or the detachment of a slough. It varies in amount all the way from a streaked saliva to a very profuse flow of blood.

Prophylaxis against hemorrhage should include (1) the correction of profound anemia when the condition is not directly traceable to absorption from the tonsils themselves; (2) the avoiding of operating on throats that have recently been the seat of acute inflammations, especially if there has been any infiltration of the surrounding tissues, e. g., peritonsillar abscesses; (3) a careful inquiry into the family and personal histories with reference to bleeding, with careful coagulation tests in suspicious cases. In this connection, it is well to remember that in spite of the statements of patients and even of their family physicians to the contrary, hemophilia is a very rare condition. Osler states that the coagulation test together with the history of repeated serious bleedings furnish the only certain evidence of its presence. In this class of cases, proper steps must be taken to reduce the clotting time to a normal period by the use of calcium salts, pituitrin coagulose, etc.

Although the case in which a careful pillar dissection has been accomplished sometimes bleeds profusely, whereas the more or less mutilated one makes an uneventful recovery, nevertheless a clean, careful dissection which leaves the pillar symmetrically intact lessens the liability of bleeding as well as greatly increases the ease with which it may be checked.

During the operation the throat should be kept as dry as possible, so that the bleeding points may be more quickly detected.

Morphin and atropin given hypodermically one-half hour before the anesthetic is begun are very useful in diminishing the troublesome flow of mucus.

Sponging is best done with the finger, instead of with sponge-carriers, so that tears in the friable pillars and soft palate may more readily be avoided. Better still is the use of the Miller pump (V. Mueller & Co., Chicago), by which ether can be administered and the throat kept dry with little or no sponging at all. I am deeply indebted to my colleague at St. Joseph's

Hospital, Dr. Joseph Z. Bergeron, who first taught me the advantage of this machine.

Slow wire snaring, in preference to the quicker cutting methods, greatly lessens the bleeding. This statement may be questioned. The almost entire absence of bleeding in using a snare, however, in cases in which the first wire has pulled out and a second one has been substituted, thereby prolonging the time that the tonsil base is held under pressure, would seem to emphasize the importance of using a slow *écraseur*.

On the removal of the first tonsil, the anterior pillar should be retracted and the entire tonsillar space examined for bleeding points, before the dissection is commenced on the other side. These may occur anywhere. If the bleeding from these points is not appreciably lessened by digital compression within a minute or two, or if it was unusually brisk to begin with, a long-shank artery-clamp must immediately be used until the other tonsil or adenoid tissue or both have been removed.

As the blood lost from the removal of the adenoid tissue forms a considerable part of the total amount lost on the table, an effort not usually made now should be put forth to minimize the bleeding from this source. An ice-pack at the back of the neck is of decided advantage. Tannic acid carried into the nasopharynx on the gauze with which the final curettement is made is often of great service. Any tags of tissue left hanging have always been considered a likely cause for subsequent bleeding. Serious hemorrhages after removal of adenoid tissue are rare indeed. In these instances, the Boettcher nasopharyngeal pneumatic tampon is of service.

The most important thing in serious bleeding is its early recognition; only in those cases where the patient has bled constantly for hours and swallowed the blood until he has become almost exsanguinated does the situation present very much difficulty. Here the outlook is grave indeed, as stated in the opening paragraph of this paper.

To avoid bleeding of this sort, or at least to discover it before any great damage has been done, the following precautions should be taken immediately on returning the patient to bed:

1. The patient should lie on the face and stomach, without any pillow, with foot of bed raised 6 to 8 inches. If there should still be a question as to whether blood is being swallowed, the swallowing reflex must be carefully watched.

2. The throat should be kept as quiet as possible. The saliva and blood should be allowed to drain from the corner of the

mouth, and not spat, hawked or coughed out. Talking should also be prohibited.

3. Food and drink should be withheld for several hours, or until all bleeding has stopped and the sputum is white. Swallowing dislodges the clots which are sealing the severed vessel mouths and fluids elevate the blood pressure.

4. Morphin hypodermics in sufficiently large quantities and administered often enough to keep the patient quiet are of great value.

5. Rapid pulse, great thirst and increasing pallor should be noted, as they are signs of impending danger. Vomiting of large quantities of black or brown blood, if it is streaked with red, or small quantities of bright red blood are the danger signals for which the nurse must immediately summon the surgeon.

If the bleeding persists after the removal of the artery clamps, or if the bleeding was uncommonly profuse before the clamps were applied, the pillars should be sutured immediately and the patient should not be returned to bed until the throat is dry.

In the event of postoperative bleeding occurring either as a late primary or a true secondary type, the patient should immediately be returned to the operating room for the pillar sutures. If the loss of blood is rapid, digital compression in the absence of tonsil hemostats is of advantage, until the sutures can be placed.

The instruments that are used in addition to ordinary long-shank artery clamps, tongue depressor, mouth-gag, pillar retractor and scissors, consist of a needle, a hook, and a pillar advancer, the latter two being mounted on the same handle. The needle is curved and is a modification of the Yankauer septum needle. The hook is small, sharply curved, and is carried on a shaft about 4 inches long, and attached to its handle at an angle of about 120 degrees. On the other end of this handle on a shaft of equal length and carried at the same angle as the hook, is the pillar advancer, which resembles the proximal part of a grooved director. Its roughened surface is of great help in working the pillar tissue over the needle point.

Silkworm gut is used as suture material, because it ties so easily and the knots never slip, as catgut would in the wet throat. Silk is not desirable from the fact that it tears the tissue so easily.

General anesthesia is useful but not necessary. With the soft palate drawn forward by the pillar retractor placed behind

the uvula, the curved needle threaded with silkworm gut is passed through the upper part of the posterior pillar, out through the lateral side of the tonsil space and forward through the anterior pillar. The bifid end of the hook is used here to work the friable tissue over the needle point. The thread is then pulled through with the curved hook. The needle is now withdrawn, leaving the suture in position to be tied. This must be done with the index finger of each hand well back in the throat, so that undue tension will not be put on the friable mucosa. The first suture now takes the place of the pillar retractor and the second suture can be very rapidly placed opposite the bleeding point. In rare cases where there is a generalized oozing, a third suture may be placed below.

With the bleeding checked, the lost body fluids may now be safely replaced by salt solutions intravenously, subcutaneously, or by rectum. The latter method can be used to advantage with either of the other two for its peculiar stimulating effect. The distressing thirst, too, may now safely be relieved by hot or cold water in small quantities. A castor oil purge the next morning does away with the febrile reaction resulting from the absorption of the digested fibrin of the swallowed blood.

On the following day, the sutures are snipped with blunt-pointed scissors and removed with forceps without any pain whatsoever. Any sort of a blunt instrument is then pressed between the pillars on each side, and the symmetry and integrity of the throat is reestablished quite as perfectly as if no complication had been encountered.

In the scholarly article of Gerhard Hutchinson Cochs, (*Med. Rec.*, June 1, 1912) he reports thirty-six cases of severe tonsil hemorrhage with two deaths. Of the fatal cases, the tonsils in one were removed with galvanocautery, in the other with a tonsillotome.

Of the entire number reported, two were done with snares, two with galvanocautery, four with scissors and knives, and the remainder with tonsillotomes of the Mathieu, Mackenzie or Bishop type.

The following measures are enumerated as having controlled the hemorrhages in the above cases: Tonsil clamps, digital compression, pressure with cotton applicator, rectal feeding, ice held in mouth, intravenous saline, transfusion, carotid ligation, suturing of pillars (in one case a pledget of cotton was sewed in between the pillars), artery clamps and ligatures, adrenalin, Paquelin cautery, astringents (alum, tannic and chromic acid,

Monsell's solution and peroxid of hydrogen) and ice-bag. In four cases the fainting of the patient terminated the hemorrhage.

In addition to these, I may mention sixteen cases from my own experience:

The ages of the patients ranged from 3 to 67 years. The bleeding began all the way from one hour to three days after the operation. In seven of the cases, the quantity of blood lost was profuse, in five it was moderately large, and in four comparatively small. The bleeding in seven cases was from the right tonsillar space, in six from the left, and in three the location is not recorded. In three cases the bleeding was from the supra-tonsillar fossa, in the others from lower down on the raw surface.

In ten cases the Mathieu tonsillotome was used, in one, scissors and in four, the snare. The other two cases were peritonsillar abscesses.

In seven cases general anesthesia was given, the remainder having been done under local anesthesia.

In three cases the pillars were sutured to check the hemorrhage, in one the bleeding vessel was caught and ligated, and in the others clamps were used.

Of the sixteen cases reported, three were seen in consultation, leaving thirteen hemorrhages occurring in thirty-five hundred operations. Of these thirteen, one was in a malignant growth and the other in a very anemic individual.

The only fatality occurred after an incision of a peritonsillar abscess by a general surgeon. The bleeding had stopped before the author arrived. In spite of the resuscitating measures that were employed, the man died the following day.

Pillar suturing is of course by no means a new idea, the Wagener-Michel metal clamps having been used for several years with varying success. The technic and instruments described may, however, be of service.

The use of sutures is a precise and definite surgical procedure for the control of hemorrhage, in bold contradistinction to the reliance on styptics and astringents as formerly practiced by even competent pharyngologists.

Pressure, either digitally or by the use of the tonsil hemostat, is of use only under certain circumstances. It is, however, always open to two very grave objections:

1. Undue traumatism is made on the tissue inside and outside of the throat, thus causing the patient much pain and sub-

jecting him to the liability of extensive sloughing in neck and pharynx as well.

2. Pressure is not the recognized surgical method for the permanent control of hemorrhage—suturing is. In the former a relatively large, indefinite area is compressed, while in the latter the exact bleeding point is caught and securely held.

CONCLUSION

The throat man is a surgeon, and his surgical problems should be handled along the line of sound surgical principles. In this way, much of the odium that has been attached by the laity and profession alike to the surgery of the throat, surgery that has had at times too much resemblance to bloody butchery, will be dispelled.

SOME OBSERVATIONS ON THE MODERN MASTOID OPERATION

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LOS ANGELES

The indications for the simple or the radical mastoid operation are not always well defined, and in consequence the personal equation of the operator has much to do with the choice of time, except in those cases with symptoms well marked.

In nearly every case of mastoiditis, either acute or chronic, we have a ruptured tympanic membrane. Many surgeons have probably operated for the cure of a chronic mastoiditis in which the tympanic membrane was intact at the time of the operation. In one case I found all the cells except one free from any evidence of disease. The one large cell was full of a dark exudate. This case probably belonged to the neurotic type, which is rare as compared with the inflammatory or infectious type.

I have had one case of acute double mastoiditis, combined with sinus thrombosis of one side and complete loss of caloric reaction of both labyrinths, with intact drums. The history was, however, that a week previously there had been a discharge from one ear.

Cases of acute mastoiditis do go on to spontaneous recovery, and many sufferers from chronic mastoiditis die from old age; nevertheless, I think it a serious error of judgment for one to treat either condition expectantly after symptoms indicate the disease. Intracranial complications are so many and so often and danger of operation so slight that one is not justified in presuming an acute or chronic purulent mastoiditis will recover without operation. If after a week in a given case of acute purulent inflammation of the middle ear there persists a slight rise of temperature, with or without swelling or tenderness of the mastoid, and pus comes pulsating from the middle ear, I think a simple mastoidectomy is indicated.

If a temperature that is at all above 100 F., persists for forty-eight hours, after spontaneous rupture or paracentesis, it is advisable to drain the mastoid. A meningitis may come on at any minute and have its origin either in the middle ear or mastoid process. Early drainage of the mastoid is the surest prevention

of meningeal complications. Preceding any surgical attack of the mastoid in which brain complication is suspected a lumbar puncture should be made and cerebrospinal fluid examined. A case of serous meningitis may recover, but from personal experience those with a diffuse purulent meningitis do not.

The one symptom in acute mastoiditis that I have come to look on as very nearly positive, with or without rise of temperature or tenderness of the process, is a pulsating discharge of pus from the middle ear. To get this distinct sign, the ear should be cleansed so that a perfect view of the drum can be had. The pulsation is an indication of tension from rapidly forming pus in the antrum and possibly the mastoid process. The more pronounced the tension and pulsation, the more positive the indication for immediate operation.

Pain and swelling of the mastoid is a variable sign. A short time ago I performed a double mastoid operation on an infant 10 months of age. In this child there was no swelling or redness of the mastoid; there had been, however, a persistent discharge for a period of three months, daily exacerbation of temperature and mild general sepsis. The absence of some post-auricular swelling in an infant so young is unusual. In the above case both lateral sinuses were covered with a soft necrotic mass and the antra full of pus.

In a few acute cases we have a slight discharge, no other general symptoms other than malaise or possibly a slight rise of temperature on exertion. In others, pain in some portion of the affected side of the head with discharges; while exceptionally we find a case with all the classical symptoms—pain, swelling, redness, tenderness, stiffness of the neck muscles, persistent discharge and pinched countenance being symptoms any one should interpret.

Blood examination should be of value in deep complications where we have a sinus complication or meningitis. Sometimes in a beginning sinus involvement blood cultures are negative. More than one blood culture should be made. A bacteremia indicates infection of the blood-stream and may be present in a case of suppurative mastoid and due to infection in some other part of the body. Clinical symptoms of a mastoiditis are far more valuable than any blood examination. Acute cases when due to pneumococcus or *Streptococcus mucosus capsulatus* infection, alone or mixed, are as a rule more fulminating and insidious than a *Streptococcus pyogenes* or staphylococcus infection.

It is interesting to contemplate why apparently typical cases of mastoiditis go on to spontaneous recovery and others require operation; why in one case the infection confines itself to the air spaces of the mastoid process and in the other spreads to the haversian canals of the squamosa. In those cases of spontaneous recovery the organism is short lived and has a tendency to confine itself to tissues of greatest vascularity.

In operative cases the organisms have a tendency to attack bone structures, sometimes blocking the blood-supply of the bone and ending in early necrosis. The morphology of the organism governs the character of the tissue change.

Mastoiditis under some favorable conditions may become epidemic and is due to the virulence of certain organisms with a predilection to middle-ear cavity and mastoid cells. In some cases I have observed, the pus from the middle ear will be filled with micro-organisms, and when the mastoid cells are opened the extravasation within contained no bacteria at all.

My one case of mastoiditis from the Klebs-Loeffler bacillus ran a very protracted course. There was much pain in the mastoid, little rise of temperature, history of previous sore throat, some tenderness of mastoid and a white, bulging drum. Thick, yellow pus followed a paracentesis. The mastoid wound was unusually slow in healing and pain persisted in the mastoid for many days after operation. Pain in the mastoid region following mastoidectomy varies in individuals and may persist for a number of days after the patient becomes ambulatory. Dry heat, codein and aspirin usually relieve the irritation.

As a confirmatory evidence of the existence of an acute mastoiditis, the value of radiography cannot be overestimated. The findings are as positive as in nasal-sinus disease and even more so. Each mastoid should be photographed separately; by doing so they can be more carefully compared. Not infrequently the extent of infection and area of the mastoid involved can be ascertained. In a case in which the operator is inclined to procrastinate or feel overconservative or in doubt, a radiograph should be made.

The symptoms indicating a radical mastoid operation are not always as well defined as those indicating the simple operation. One symptom, however, stands out preeminent and that is a chronic, foul discharge from the ear. A foul discharge is indicative, as a rule, of caries, necrosis or cholesteatomata. It may be taken as an axiom that in all cases of a chronic discharging middle ear, regardless of the character of the discharge, a

meningitis is always a possibility. Other indicative signs are labyrinthine symptoms, periodical or constant pain on the affected side, and exacerbation of temperature.

In many cases, the pathologic process is confined to the middle ear and antrum and may be relieved by curettement, ossiculectomy or medication. These cases are the exception and the treatment cannot be other than empirical. With no indications to the contrary, it is a safe rule to do the radical operation in all cases of chronic discharging ear.

There are only two or three conditions that may contra-indicate a radical or semiradical operation in a chronic middle-ear and mastoid suppuration. Patients in an advanced stage of pulmonary tuberculosis usually die, following either a simple or radical operation. The vitality of the patient at such times is too low to withstand the shock of the anesthetic or repair the wound. Infusion anesthesia may be indicated in such cases rather than the American or drop method.

In preparing the patient previous to the operation, more care should be observed in women than in men, on account of the greater tendency to gastro-intestinal disorders, especially disease of the colon.

In the preparation of the site of the operation, it is needless to shave the side of the scalp in proximity to the line of intended incision. We should try to preserve the hair, and not render our patients unnecessarily conspicuous. The healing of the postauricular wound usually takes from one to five weeks, long before the hair, if clipped or shaved from the scalp, reaches the length worn by the patient.

It is a custom with me to paint the pinna and auditory canal and postauricular region well into the scalp with tincture of iodine. If the case is seen the day or a few hours previous to the time set for operation, the iodine solution is then applied. When the case comes to the operating table, if the skin is not unusually sensitive, the iodine solution is again applied. This is the only external preparation. Scrubbing, shaving, and bichlorid packs, I think are unnecessary.

To prevent the hair from getting into the wound at the time of operation, strips of gauze are cemented along the edge of the scalp with collodion. These strips are easily removed after the operation is completed, by moistening with ether. After the collodion strips have firmly adhered, sterile towels are bound about the head.

The auditory canal is now, as far as possible, cleansed by irrigating with a warm bichlorid solution (1:5,000). Afterward, the canal is filled with pure alcohol and plugged with cotton.

The anesthetic used is invariably ether, preceded sometimes one-half hour in extremely nervous adults by $\frac{1}{4}$ grain morphin and $\frac{1}{150}$ grain sulphate of atropin. Ether is administered by the American or drop method. On two occasions I have given ether by intravenous infusion, and with happy results. The solution used was 7 per cent. ether and 93 per cent. normal salt solution. We do not like gas-oxygen anesthesia and this is not said to disparage those who favor this method of anesthesia. Our opinion of this form of anesthesia has been influenced by two nearly fatal results, and bad results frequently observed in medical literature. In an experience of over fifteen years, we have had but two deaths from ether anesthesia. Post mortem disclosed in one a typical case of status lymphaticus. We have exercised, however, the greatest care, and only a few times have we ever trusted a case to a man who has not had some experience in the administration of anesthetics. It is only in the beginning that a deep anesthesia is necessary. If the anesthesia has been a prolonged one, we sometimes use oxygen after the operation is completed to hurry the elimination of residual ether in the lungs. A light anesthesia and rapidity in operating is the *sine qua non* of a mastoid operation. The less shock, the more rapid the recovery.

Speed, consistent with safety, is essential to success. The antrum is reached, we think, more quickly with the gouge designed by Alexander and driven by a mallet. The chipping away of bone should be begun with the largest sized gouge and in the direction of the spine of Henle, and as we approach the antrum a smaller gouge may be substituted. In the hands of the skilled, the operation on the bony structure may sometimes be completed with a mallet and gouge. We should in this operation, or in any other, try to simplify our armamentarium. In the simple mastoid operation and after the cells are completely removed, the posterior bony wall of the auditory canal should be shaved down nearly to the annulus tympanica. In very young children this is not indicated. In adults the healing is simplified by allowing the membranous posterior wall of the auditory canal to drop back into the wound. Previous, however, to prevent deformity, as much of the external wall of the tip is saved as is consistent with thorough exposure of all the

tip cells. The wound previous to closure is thoroughly cleansed with peroxid of hydrogen, followed with alcohol. Afterward, the periosteum and subcutaneous structures are firmly united with a No. 2 plain catgut suture, except near the inferior angle of the wound. By careful attention to apposition of subcutaneous structures we avoid, in most cases, a depressed scar. At the inferior angle one or two stitches are usually taken, leaving an opening near the most dependent portion of the mastoid wound, not larger than one-half inch, through which a strip of 5 per cent. iodoform gauze drain or small cigarette drain is gently passed into the wound in the direction of the aditus ad antrum. Sometimes the gauze is softened by dipping in warm vaselin, which prevents adhesion to the exposed bone and tissues.

We can see no good reason for firmly packing the bony cavity. There is a tendency among some operators to pack the wound too tightly and for a longer period of time than necessary. The less packing the smaller the scar and more rapid the recovery. After the cells are removed, the abscess cavity has a tendency to take care of itself and therefore any artificial drainage is to be avoided. The tendency of a mastoid wound after the cells are drained is to go to spontaneous recovery if we will leave the wound alone. The cutaneous structures are closed with Michel's clips in patients past the first year of life; if in an infant, the lips of the wound are closed with a ten-day chromic catgut. In older children and adults Michel's clips may be removed in from twenty-four to thirty-six hours. The sooner the clips are removed the less likelihood there is of a scar.

The external surface is now cleansed and a layer of gauze saturated with pure alcohol applied and over this the usual dressing. In the great majority of our cases we have no post-auricular depression and very little scar at the line of incision in either the simple or radical operation. A blood-clot operation has nothing better to offer except, in a few favorable cases, a more rapid healing.

If the operation is performed in the morning, and on those past infancy, the external dressing is removed in the evening and a new dressing applied. Daily and sometimes twice daily the dressing is removed, the auditory canal irrigated with warm 1:5,000 bichlorid of mercury solution and a warm, moist dressing applied about the ear and covered with dry gauze. By taking this precaution we save the patient a great deal of pain and hurry the recovery many days.

The patients are always pleased with frequent change of external dressing, pain is greatly relieved and better sleep assured. The patient is usually up and about on the fourth day. As soon as the gauze strip comes away moistened with serum or slightly tinged with pus, we permit the wound to close. It is unnecessary for the wound in the bony canal to be filled with granulations before the external wound is allowed to close. The treatment of the postauricular wound following the radical operation varies but little from that described above. As to the form of the meatal flap, we more often use the Jansen, though sometimes the Körner flap. We do not trouble ourselves with skin grafts.

After a week I try to discard the bandage. The wound is covered with a strip of gauze and cotton half-moon shaped, which fits behind the pinna and is held in position by collodion. If the posterior wound is slow in closing, we pass a cotton-tipped probe saturated with phenol as far into the fistulous tract as it will go. One application of this is usually all that is necessary. A postauricular fistula may persist for a long time and try the patience of Job before it is cured, but fortunately such a case is rare.

Sometimes where necrosis has been of long standing, or the catgut fails to be quickly absorbed, healing is retarded by the formation of excessive granulations about the mouth of the wound. Cutting them away does not always stop their recurrence, neither does the application of escharotics prevent their growth. These cases are exposed to air and sunlight as long as possible, the patient often going a whole day with the wound exposed. In winter the dry air of the house suffices and in summer the free warm sunshine is sufficient to dry the granulations. It is remarkable how quickly these cases recover after the bandage is removed and the granulations exposed to the air and sunlight. The value of heliotherapy is given but little consideration by otologists, and a treatment so simple and natural should be applied more often. Air and sunlight are very essential and efficient in healing the middle-ear cavity after the radical as well as the simple operation. In the radical operation, as soon as the patient becomes ambulatory, all dressing should be removed from the canal and the ear exposed for as many hours as possible to the rays of the sun. All forms of dressing retard repair. There is practically no danger of any new infection when the reparative process has once begun.

A patient should not ordinarily remain longer than a week in the hospital for a simple or radical operation. The expense while there to the average individual is very great and sometimes eats up most of the money that would come to the physician. As long as the patient is free from rise of temperature and able to be out of bed, there is no good reason why he should not report daily at the physician's quarters for dressing. Catgut sutures are a potent factor often in retarding recovery. The longest period I recall of plain catgut remaining unabsorbed was eight weeks.

As to the use of urotropin, we have given this drug in most cases of severe infections of the mastoid in which we suspected a possibility of some brain complication. There is sufficient literature on the subject to prove that the spinal fluid contains urotropin when administered per os to encourage one to use the drug. In young children and even adults the irritation to the kidneys is very great when the drug is given in large doses. In infants the urine will be tinged with blood and in adults great pain will be experienced on urination.

As to vaccines, I have come to believe in them after operation and not before. I have had what appeared positive results from vaccines in acute mastoiditis, only to have a second attack, fulminating in character, come on a few weeks after the ear was apparently cured. Where the antrum was exposed four to six hours after the second attack it was found to be filled with debris. I am rather skeptical about the efficiency of vaccines in suppuration in closed cavities, such as the nasal sinuses and mastoid antrum and process. If the case is fulminating and one organism is present in the pus from the canal, I give the vaccine at the operating table; if more than one organism, I give the mixed vaccines which are known as stock. If the case has been under observation for sufficient time, I prescribe autogenous vaccines. In the great majority of cases we do not have time for the preparation of autogenous vaccines. Even had we time, our faith in stock vaccines prepared by the Lederle Laboratory in the east or the Cutter Laboratory in the west is greater than in an autogenous vaccine prepared by some one who is not a specialist in such therapy. It would be better in the preparation of vaccines to secure the pus from the mastoid wound rather than the auditory canal, and in virulent cases from the bone chips as well as from the free pus in the mastoid. The vaccines help to prevent new infection and hurry repair. One dose is usually all that is given.

The accidental exposure of the dura or lateral sinus in the simple or radical operation, as far as I am able to judge, is free from danger. It is reasonable to presume that in the exposure of the dura or sinus we avoid cutting or tearing the tissue. A meningitis or phlebitis, if it occurs, has had its inception before operation. The dura has a very strong natural resistance to infection, and this is much greater after free drainage is established. Meningitis and thrombosis are diseases with a predilection to precede rather than to follow a mastoidectomy. If we have any suspicion of the possibility of the sinus becoming involved a free exposure of the sinus is made.

The so-called Heath operation has many points to recommend it to the otologist. It is a reasonable presumption that the removal of the posterior bony wall of the canal down to the annulus tympanica, with the formation of a flap, for the cure of acute mastoiditis is not an unsurgical procedure. By so doing we avoid filling a deep cavity with fibrous tissues, prevent the recurrence of mastoiditis, and secure more rapid healing. In the few cases in which I have used this method the results have been perfectly satisfactory. The one disadvantage of all flap operations is the tendency to accumulation within the canal of cerumen mixed with debris. Very often these cases require attention at intervals for many years.

In the consideration of the radical mastoid operation, the preservation of the ossicles, tympanic ring and some of the tympanic membrane as advocated by Heath, Boudy, Ballenger, Botey and many others has given me, as it has most operators, a good deal of concern. We are idealists and are looking for ideal results. It remains a question of dispute as to the advisability of trying to retain the ossicle and tympanic ring and probably the question will be discussed for years to come. It is impossible to tell anything of the necrosis of the ossicles until they are removed. Since the incus and some part of the malleus are usually destroyed and the chain of articulation broken in all chronic suppurations of the middle ear, the removal of the necrosed ossicle does not jeopardize the hearing. In many cases the malleus is so broken down by adhesions that its function is completely lost, and its removal may benefit the hearing. The size and shape of the drum may govern the indication for or against any effort to retain the tympanic ring and ossicles. Where the tympanic membrane is destroyed and the antrum filled with cholesteatomata it is best to do the classical tympanomastoid operation. If as far as the eye can detect the ossicles

are intact, the drum membrane not in apposition with the inner tympanic wall and there are no symptoms that would indicate the possibility of a labyrinth irritation or meningitis, an effort may be consistently made to save the drum membrane and ossicles.

I presume I am subjecting myself to a good deal of criticism when I say that I seldom pay any attention to the orifice of the eustachian tube in the radical mastoid operation. The tube as a factor in prolonging any suppuration after radical mastoid operation is, I think, overestimated. The cause of any failure to secure a dry ear is, as a rule, outside of the eustachian tube. Speaking advisedly, I regard it as a disease of the bony structure which surrounds the antrum, and particularly along the aditus ad antrum, which retards recovery.

STATISTICAL TABLE SHOWING TREATMENT AND RESULTS IN DR. KYLE'S MASTOID CASES

Mastoiditis, acute cases, operated	227	
Mastoiditis, chronic cases	76	
Mastoiditis, T. B., advanced pulmonary lesion	3	
Mastoiditis, T. B., advanced pulmonary lesion, operated	3	
Mastoiditis, T. B., advanced pulmonary lesion, deaths	3	
Mastoiditis, Bazold	3	
Total		309
Meningitis, otitic origin, purulenta diffusa	11	
Meningitis, following acute mastoiditis	7	
Meningitis, following chronic mastoiditis	4	
Meningitis, recoveries from purulent meningitis	0	
Meningitis, localized	3	
recoveries	3	
Meningitis, circumscribed purulent	5	
recoveries	5	
Sigmoid sinus diseases, thrombosis	10	
Sigmoid sinus disease, deaths	4	
Sigmoid sinus disease, ligated	5	
Sigmoid sinus disease, recoveries after ligation	2	
Sigmoid sinus disease, phlebitis	10	
Sigmoid sinus disease, recoveries	10	
Total		25
Brain abscess, temporo sphenoidal	1	
recovery	1	
Brain abscess, cerebellar	1	
Brain abscess, deaths	1	
Extradural abscess	5	
Total		7
Paralysis facial, acute mastoiditis	2	
Paralysis facial, recoveries	2	
Paresis fascial, following operation on chronic	5	
Paresis fascial, recoveries	4	
Total		7
Labyrinthitis, hyphremia	11	
recovery	11	
Labyrinthitis, suppurative, operated	2	
recovery	1	
Labyrinthitis, serous	4	
Labyrinthitis, perilabyrinthitis	5	
Total		22
Mastoid cells opened by mistake	4	
Angioneurotic edema	2	
Angioneurotic edema, opened by mistake	1	

July 24, 1914.

HARMLESS POST-OPERATIVE TEMPERATURE

GEORGE F. COTT, M.D.

BUFFALO

The danger of chronic suppuration of the middle ear is apparent to all and I would not like to detract one iota from the value of the symptoms indicating that danger lurks near. We often have typical lesions, while at other times the lesion is altogether out of proportion to the symptoms. A brain abscess may be latent for an indefinite time without any symptoms whatever, but usually there is some indication of hidden trouble. Occasionally multiple abscesses occur when but one is suspected. Pachymeningitis is frequently found, having manifested no apparent symptoms, and even epidural abscess may be discovered occasionally during operation. These cases may be exceptional but they occur with sufficient frequency to make one careful before giving an off-hand opinion.

In pachymeningitis with pus, we expect pressure pain with perhaps intermittent discharge; the pains may be very severe, mild or absent altogether. In leptomeningitis we have irregular temperature, stiffness of the neck muscles, Babinsky, tache cérébrale, Kernig's sign and other nervous signs, which may be augmented by examination of the spinal fluid. Brain abscess usually gives us a slow pulse, dull pain, low temperature, slow mentality, sleepiness or somnolence. If the abscess is located in the cerebellum, we would usually have in addition nystagmus toward the side of the disease; here also would be dizziness and occasional vomiting. In phlebitis, especially of the lateral sinus, which is most affected in otological cases, there is commonly the steeple-peaked temperature of slow or rapid repetitions according to the virulence of the infection. In septic thrombosis you have in addition to violent chills and sweats, depression from sepsis and a high leukocytosis. In suppuration of the internal ear there is dizziness, nystagmus, often vomiting and deafness, following caries of the middle ear.

The case becomes more complicated when several of these lesions occur together and then of course disaster is more apt to follow. Yet nature is very kind to some patients. I have seen a man walk from his bed to the operating room when a radical operation was done, manifesting no special symptoms but

enough pain to cause him to consult his physician. On his death the second day after, we found one entire hemisphere covered with pus, which trickled on the floor as the upper calvarium was removed. He was an old syphilitic. In another case, a lady walked into the office with pus discharging into the external auditory canal from a brain abscess. A third was sitting in a chair, temperature 104, some pain deep in the ear, but refused at first to go to a hospital, not feeling particularly sick. At operation the same day, we found a circumscribed epidural abscess, a thrombus in lateral sinus, which had broken down and formed pus in the sinus.

These are unusual cases but they may occur at any time; and with caries of the tympanum should always be suspected and

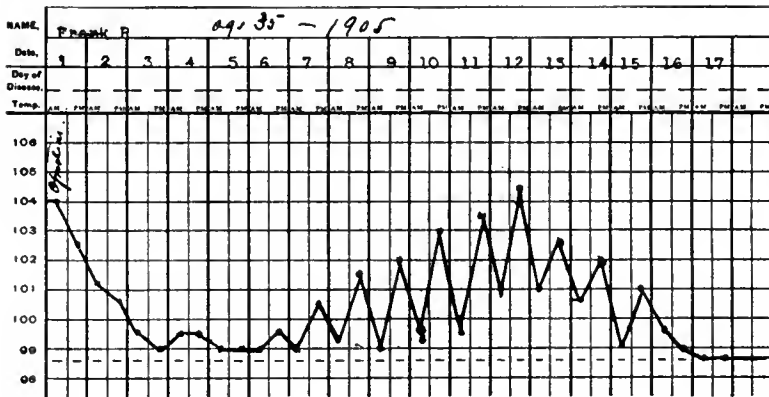


CHART I, CASE 1.—RADICAL MASTOID OPERATION

Sinus thrombosis, which had broken down and formed pus in center; epidural abscess; after operation temperature fell to normal, after the seventh day temperature rose and continued to rise and fall for a week, then gradually recovered. At the height of the fever, which reached 104.5°, the consulting surgeon said the patient would die, with which I disagreed. Recovered.

never passed over lightly. Unless one sees a number of such cases with brain complications, one is apt to overlook some common symptoms and the patient may succumb; on the other hand some really prominent symptoms may mean very little but may impress the physician greatly and cause unnecessary worry. This is more apt to occur in small towns and in country practice, where the attending physician is solely dependent on himself. The surgeon is often called to operate out of town, not knowing much of the patient's history and acting only on present indications, then not seeing the patient again. Disaster occasionally follows the operation because not enough was done; still the patient may have gone under even if the exact con-

dition had been ascertained. However, a doctor's conscience is more at ease when he is fairly sure of the status of the patient. If these patients could be observed a few days before and after the operation, one might be able to render better satisfaction. Often operations become necessary when the physician makes his first visit; it is unfortunate, but sometimes circumstances may require immediate action.

One important item must not be overlooked. It is not at all uncommon for patients to deny any former ear trouble. Brain affection due to the ear is at once dismissed and an opportunity to save life has thus flitted away. A number of such cases on examination were found with scars on the drumhead, or old perforations, leaving a good field for developing bacteria. In

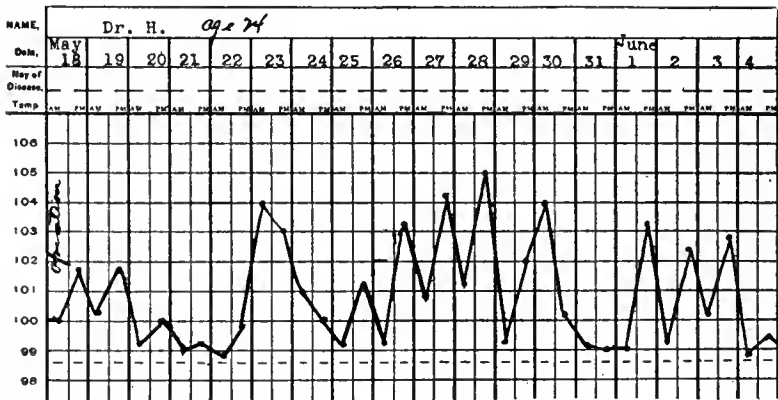


CHART II, CASE 2.—RADICAL MASTOID OPERATION

Caries; fifth day after operation temperature rose and continued to rise and fall for a week. At the end of that time, in consultation with two eminent men, an internist and a surgeon, it was decided to explore the lateral sinns. But the next day the temperature returned to normal and I put off operation; final recovery.

all suspicious cases the ears ought to be examined before one dismisses them as a factor in meningeal or brain infection.

I would like to call your attention to a procedure of importance which any man can carry out and which will always give him fairly accurate information, and that is the examination of the blood.

If you find a moderately high leukocytosis with a normal polynuclear percentage followed in ten or twelve hours by a mild recession, the next day and the following day the same, you may be quite sure your patient has no complication and will speedily recover; if this condition is reversed, look out for breakers ahead; and this shows twenty-four to thirty-six hours before any other symptoms are noticed. If, for instance, your

patient runs a recessional temperature of 106 or more and the leukocytes are normal, there is nothing to fear from infection. On the other hand, if the temperature is normal or below and your leukocytosis is high, say 20,000 or 25,000, there is something brewing, and rapidly too. There are four conditions in which the temperature could be below normal in serious cases, and that is in brain abscess, sinus thrombosis, shock and approaching dissolution.

If the temperature is taken every four hours and a blood count made twice a day where any complications are suspected, one can act far more intelligently than where these things are omitted. Especially is it true with septic sinus phlebitis. You may find the temperature 106 or below normal at your daily

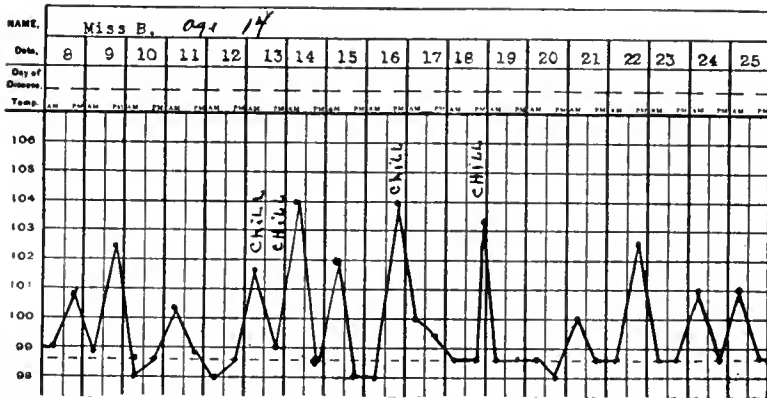
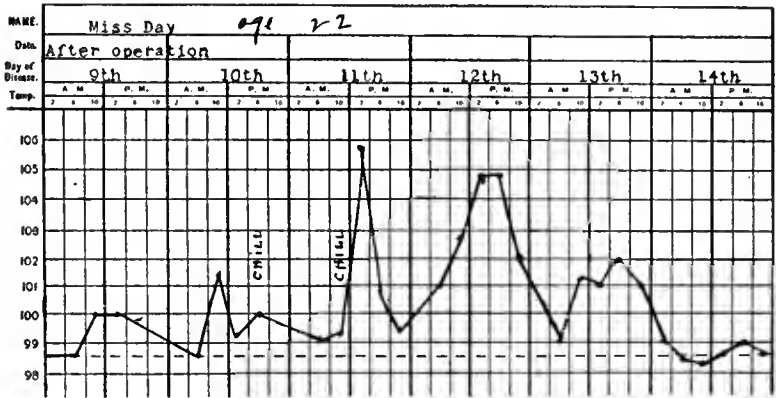


CHART III, CASE 3.—MASTOID DISEASE

The peculiar temperature caused an examination by her physician and a surgeon, and they concluded she had endocarditis. The only murmur heard was over the left scapula towards the vertebrae. Recovery.

visit whereas had the temperature been taken frequently it would have been found that both extremes occurred the same day. Anyway the blood count would have disclosed the approaching danger. Suppose that several weeks after an operation or after an acute attack of some kind, perhaps an apparently simple sore throat, a patient gets a sudden high temperature preceded by a chill, sometimes of considerable severity, lasting for several minutes; and suppose these chills agitate the patient so violently that the bed trembles, the temperature rising thereafter and at times receding in a few hours; at other times remaining stationary a few hours then declining slowly, and suppose that process is repeated at intervals of a few days to a week or more; with pain probably present in some distant part of the

body, with apparently no connection with the lesion in the throat or ear; in such cases there is undoubtedly infection localized somewhere, remote from the original disease. This is peculiarly the case with embolic infection. The immediate effect is a rise in temperature for a short time only. The leukocytosis may be disturbed and run to a height of 20,000, polynuclear percentage up to near the limit, in one case as high as 98 per cent., more often not over 90 per cent. Such patients may have considerable pain, or the temperature may rise, which, however, gradually subsides until another spot becomes infected. These areas of infection take place anywhere—liver, spleen, brain, lungs, joints, etc. A common cause for such abnormal temperature follows



* CHART IV, CASE 4.—MASTOID DISEASE

Caries, ninth day temperature began to rise. She had two chills, the second one lasted seven minutes, so severe that she caused the bed to tremble. No untoward symptoms except the abnormal temperature; recovered.

endocarditis, and this condition is not always easily made out. I believe it is oftener found after operations. However, the rule is that these patients usually recover. When this condition occurs early after an operation or after some septic disease, and the patient does not improve rapidly, streptococcus or *Diplococcus capsulatus* is the probable cause and the patient succumbs. The blood count will usually indicate the severity of the infection. These patients can often be saved if the diagnosis is made early. They usually recover when a distant vein is affected. The same condition exists in endocarditis. Unless the infection is carried to some other part of the body, no especial difference is noticed in the temperature; but should an embolus affect any other organ, the temperature shoots up 3 or 4 or 6 or

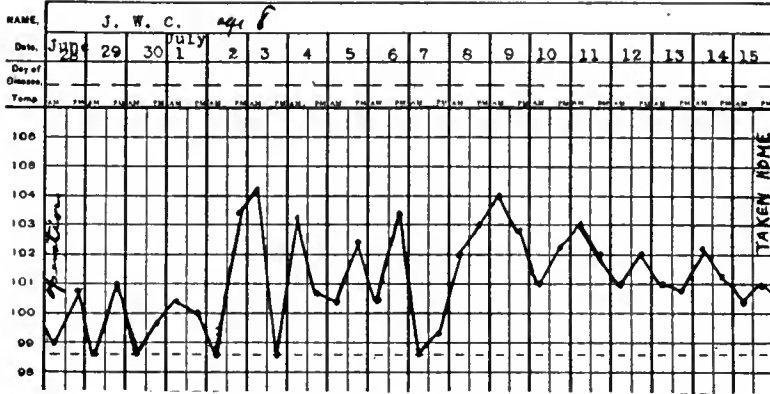


CHART V, CASE 5.—RADICAL MASTOID OPERATION

Patient thought to have typhoid fever; after two weeks drum head ruptured and discharged pus for about ten days, when I saw him. His trouble was supposed to be acute mastoiditis. Found sclerosis due to recurrent attacks; mother denied previous ear disease, but acknowledged boy had complained of earache off and on. Red cells, 4,250,000; white cells, 10,000. Lingered for several weeks after he left hospital, then recovered.

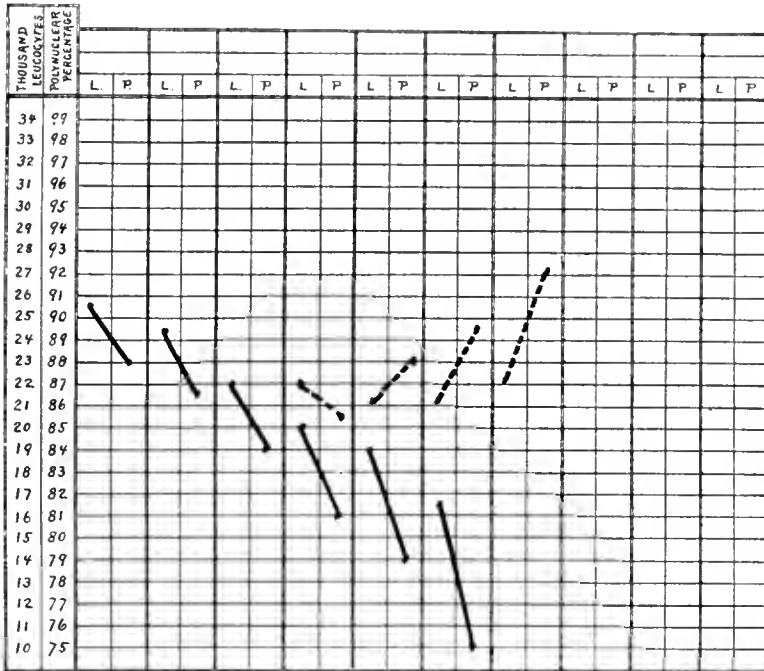


CHART VI

Broken line — Complications. Solid line — Favorable course.

even 8 degrees. If the patient ultimately recovers the temperature recedes rapidly and the constitutional effect is not marked.

The temperature in each one of my cases was postoperative, but the same occurs in any kind of infection except where there is much shock. We notice the high and low temperature in septic sinus thrombosis, but we do not know about the thrombus until this peculiar temperature occurs. So a patient is liable to succumb before a diagnosis can be made. A marasmic thrombosis usually causes no such temperature nor does a thrombus anywhere in the body where it is thoroughly organized.

A similar condition of affairs exists in infection following tonsillitis, arthritis (commonly called rheumatism), pneumonia, periotitis and bone necrosis or abscess, remote from the throat lesion. But the infection, seldom traced to the throat, since it comes on several weeks after the throat has apparently recovered, is nevertheless due to the lymphatic absorption and final distribution to parts at a remote distance. This explains many affections hitherto treated as idiopathic diseases. Furthermore, where lymphatics are abundantly distributed the presence of infection, even though mild, is rapidly manifested. This is the case in sinus disease, which may cause a rapid rise in temperature and a lymphangitis of the brain with very severe pain. A more palpable illustration is found in septic deposits, as in the heart or sinuses or even after abdominal operations. The sinuses of the brain give splendid opportunity for this demonstration.

When a septic thrombus forms, there are no other symptoms than those commonly found in any other infection, but where a particle is carried into the circulation there will be a sudden rise in temperature commensurate with the degree of infection, and every time such embolus has made its call in a remote part of the body we get a repetition of the high temperature.

As these points of hesitation are small the temperature remains high but a short time and commonly recedes in a few hours, to recur again and again; it is one way nature has of letting us know the condition of the patient, and many lives have been saved because of recognition of this symptom.

The peculiar temperature shown in the charts are all due to chemical or organic emboli and all recovered as predicted, for the prognosis is always good when the constitutional symptoms are mild, regardless of the high temperature.

DISCUSSION

DR. OTTO GLOGAU, New York: The rise in temperature following aural operations is an interesting but not rare phenomenon. It may be due to the direct or indirect irritation of the adjacent brain tissues. For several years I have given, as a routine procedure in every case of mastoid operation, whether complicated or not, $7\frac{1}{2}$ grains of hexamethylentetramin every four hours for about one week. Since that time I have found almost no postoperative rise in temperature.

EXHIBITION OF INSTRUMENTS

A NEW ELECTRIC OPHTHALMOSCOPE

CHARLES H. MAY, M.D.

NEW YORK

Among the essential improvements in this new electric ophthalmoscope are superior illumination of the fundus, and the replacement of the customary fragile mirror by a solid piece of glass which acts both as a condenser and as a reflector.

The salient features of the instrument are as follows: The metal filament lamp enclosed in the handle is a very small one (volts 2.75, ampere 0.1); this is sufficient on account of the optical construction of the instrument. The rays emanating from the lamp are made less divergent by a series of convex lenses placed immediately above, and are conducted into a solid rod of glass which acts as follows: The lower end of this refracting and reflecting device is convex and serves as a convex lens; the beam of light then strikes the upper posterior portion of the glass rod, which is here ground at an oblique angle to form a prism; this surface is silvered and acts as a plane mirror, reflecting the rays so that they enter the eye of the patient. This combination condenser and reflector, enclosed in metal, excepting in front, is attached to the anterior surface of the lens disk of an ophthalmoscope in such a manner that the upper extremity covers the lower half of the sight hole; the upper half is left free, and through this semicircular aperture the eye of the observer receives the rays reflected from the illuminated portion of the background of the eye under examination. The distance between lamp and reflector can readily be increased or decreased, thus varying the degree of divergence of the rays.

A rheostat revolving upon the axis of the stem of the instrument serves to regulate the intensity of the illumination; this rheostat can easily be pushed up or down and then serves as a switch for turning the current off or on.

The writer has utilized his double-disk instrument (*Annals of Ophthalmology*, January, 1900), in which the lenses are inter-

posed in the sight hole by rotating the milled edge of an anterior disk for the convex lenses and a posterior disk for the concave lenses; with these there is no necessity of rotating a quadrant into place when fractions of diopters or when strong



Single disk Ophthalmoscope
with Battery Handle



Double disk Ophthalmoscope
with Conducting Cord

May's Electric Ophthalmoscope.

lenses are required. The disks are very thin: thus a minimum of distance separates the eye of the observer from that of the patient—a very important point. The apertures of plates and lens openings are so arranged that they are larger than the sight hole, the edges of which are sharply beveled—thus there can be only one edge capable of giving rise to a reflex. Most

ophthalmoscopes now manufactured are poorly constructed in this respect, and as soon as the blackening is worn off, as happens after a short period, there are reflexes, which are very annoying and interfere both with the ease of examination and the clearness of the fundus picture.

The instrument is also made in simpler form, consisting of a single disk with fourteen lenses, a number sufficient for the requirements of the general practitioner.

The source of light is a small two cell dry battery contained in a handle. The purchaser may choose the size which he prefers. The lamp can be operated with a comparatively small dry cell on account of its low current consumption. By means of an adapter, the ophthalmoscope can be attached to any ever-Ready flashlight having a miniature lamp thread by removing the lamp, screwing in the adapter and fitting the ophthalmoscope in place.

The instrument can be used as a transilluminator.

The battery handle can be detached and used as a storage battery connected with the ophthalmoscope by cords; this is often convenient when examining the fundus in patients lying in bed, especially infants, under which circumstances the battery handle is often embarrassingly in the way; or the cords can be attached to the outlet from the street current, protected by a suitable rheostat.

Finally, in case the battery gives out or the mirror burns out—and this happens at unexpected times with the best electric instruments—and renewals are not at hand, the disk portion of the ophthalmoscope can be separated, readily attached to another handle with a tilting mirror, and thus converted into an ordinary reflecting ophthalmoscope for use with any convenient source of illumination.

The advantages claimed for this new electric ophthalmoscope are: (1) simplicity; (2) small size, so that the instrument can easily be carried in the pocket; (3) absence of the usual fragile reflecting mirror; (4) superior illumination, with absence of reflexes and shadows, making the fundus examination remarkably easy and giving an exceedingly clear view of the background with both the direct and the indirect methods; (5) adaptability for battery handle, any flashlight, or cords; and (6) the ability to convert the electrically lighted instrument into an ordinary reflecting-mirror ophthalmoscope when the battery is exhausted or the lamp is burnt out and renewals of these parts are not at hand.

PROTECTIVE GLASSES

NELSON BLACK, M.D.

MILWAUKEE

Dr. Nelson Black exhibited some of the glasses used in various industries for protection against intense light, and demonstrated the absorption properties of each. The harmful radiation in certain industries were described and the lack of protection with the glasses actually in use was demonstrated.

AN INSTRUMENT TO MAKE A LEECH-BITE
INCISION TO PRODUCE FILTRATION
FOR GLAUCOMA

FRANK TODD, M.D.

MINNEAPOLIS

The instrument I herewith present was made two years ago, but has never been shown. It is an instrument for the purpose of securing filtration from the anterior chamber under the conjunctiva for glaucoma, similar to the Elliot operation. My experience with the Elliot trephine operation is limited to fifty-nine cases, but even this experience has led me to believe that there are certain cases in which a lesser operation producing a smaller opening would be better.

Previous to performing the above-mentioned operation, I performed several Herbert operations, with which you may be familiar. This consists of making a tongue flap of the sclera which hinges at the corneal margin, filtration occurring through the incision thus produced. This operation is done with the ordinary cataract knife and is very difficult. Because of its difficulty I devised this knife, which makes a leech-bite incision, i. e., three lines proceeding from a central point, thus producing three flaps, an incision very difficult to close, as all of you realize if you have ever attempted to suture such an incision. Yet, while this incision remains open and allows satisfactory filtration under the conjunctiva from the anterior chamber, no scleral tissue is removed, and weakening of the sclera does not take place; so that there is less liability to protrusion of the ciliary body into the opening than where a large opening is made, as with the trephine, and a portion of sclera removed. Any one who has had much experience must have seen cases in which the ciliary

body filled the round opening made, and in which more or less disaster occurred later, because of this accident, a result which is not as likely to take place if the incision is small. Furthermore, in some cases in which a trephine opening is made, a large swelling results, being caused by the presence of aqueous humor underneath a too thin and fragile conjunctiva. It is in such cases that ultimate erosion and weakening of the conjunctiva is to be feared, and the possibility of remote infection exists. Such a bleb of conjunctiva does not occur in the case of an operation made with this leech-bite knife.

In performing the operation with this instrument a conjunctival flap may be dissected up or the knife may be shoved under the conjunctiva well above the margin of the cornea. The point of the knife may then be slid along between the conjunctiva and sclera until it reaches within 2 or 3 mm. of the cornea when it should penetrate the sclera and enter the anterior chamber, being as quickly withdrawn. If prolapse occurs, iridectomy is performed.



The operation is very simple and may be very quickly performed. It is not intended to suggest that this operation should take the place of the Elliot trephining operation, which I believe to be an excellent operation, but I am coming to the conclusion that it is not suitable for all cases, as above suggested, and that the operation herewith presented is, in certain cases, safer.

I herewith append a report of a case illustrating these points.

Mrs. L. B., aged 64, was referred to me May 22, 1913, through Dr Macleish of Los Angeles. She had been under his treatment for chronic simple glaucoma, and he advised her to have an operation as soon as she returned home.

May 22, 1913, vision in right eye, counting fingers at 1 foot. Field small and confined to temporal side. May 22, 1913, vision in left eye, 6/18. Field much reduced. Cupping of disks, right eye 5 D., and left eye 4 D. Right eye affected for five years, sight gradually growing poor. Left eye, patient had noticed disturbance for two months. Pupils seem dilated.

On May 27, 1913, leech-bite incision was made with the knife described. No prolapse of iris occurred, but iris was accidentally cut at the cornea-scleral junction producing slight iridodialysis and no prolapse. Good surgical recovery.

July 18, 1913, vision in left eye, 6/12. Oct. 15, 1913, vision in left eye, 6/12. Tension right eye 48 mm., left eye 23 mm. Filtration evident by appearance, but conjunctiva appears strong and not much elevated.

Dec. 2, 1913, Elliot trephining operation on the right eye combined with an iridectomy. This operation resulted in reduction of tension and the formation of a large bleb extending around and above the upper half of the cornea. The conjunctiva is very thin and looks as though it might readily rupture. This rupture, however, has not occurred, and when examination was last made, July 13, 1914, the tension was, right eye 3 mm., left eye 18½ mm. Vision right eye, count fingers 2 feet, field improved. Vision left eye, 6/9, field improved.

It will thus be seen that results were good in both eyes, but the very thin conjunctiva leaves a weakened swollen bleb on the trephined eye, whereas in the other eye, where filtration is sufficient, there is a strong scar in which there is no fear of future rupture though tension is normal and vision has improved.

INSTRUMENT FOR KERATOTOMY

G. B. JOBSON, M.D.

FRANKLIN, PA.

A set of instruments, consisting of a keratome, special fixation forceps and small tenaculum was devised by me for the removal of corneal scars and opacities, when situated on the surface of this structure. And the technic was described in *The Ophthalmic Record*, July, 1912, under the title of "Keratotomy." I find the keratome useful in other operative procedures about the eye, as in detaching the head of a pterygium from the cornea, or in prolonging the base of the conjunctival flap to the cornea, where it may be loosened from the cornea, by a second keratome with a dull edge, sufficiently to allow of the use of the trephine. The blade of the keratome is attached to the shank in such a way that it can be used to cut in any direction or at any angle without obstructing the view of the field of operation.

THE BINAURAL TELEPHONE

E. E. HOLT, PORTLAND, ME.

This instrument, for testing the hearing power for the voice and detecting whether deafness exists in one ear or not to the extent alleged by the patient, Dr. Holt had made primarily for the purpose of testing soldiers or sailors who applied for a pen-

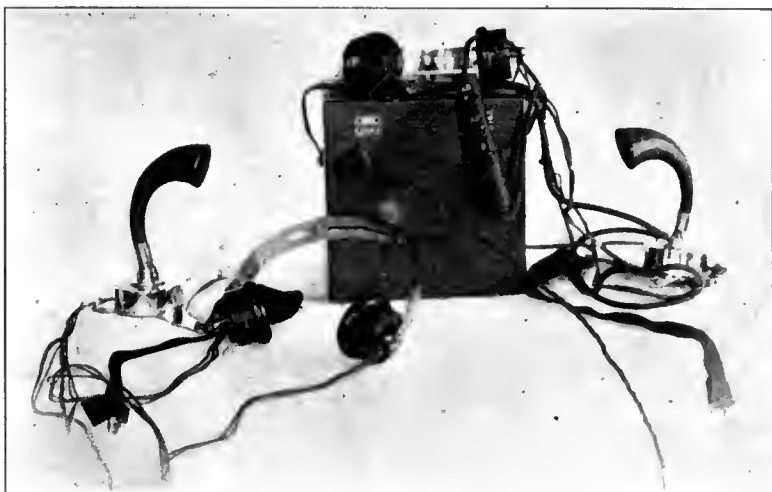


Figure 1



Figure 2

sion or an increase in a pension on account of deafness or an increase of deafness. With the ordinary tests for alleged deafness in one ear he had found it very difficult to arrive at a satisfactory conclusion as to whether loss of hearing existed to the extent claimed by the person. He therefore had this instrument made, which is shown in the accompanying photograph. He had found it not only useful for the purpose it was intended, but also in testing the hearing power of patients in aural practice generally in order to make a more definite record of that condition and the improvements obtained by the treatment pursued.

CASE BOOK RECORD

Dr. E. E. Holt of Portland, Me., presented a sample of a case book for recording cases of the eye, ear, nose and throat; it being the last edition of one devised forty years ago. These record books are made for recording five hundred cases. They are fifteen inches long and eleven inches wide. The right-hand page provides for making a complete record of the case. A large number of words used constantly in making records are printed with a square at the left of them so that a cross placed within the square gives all the desired information. There are diagrams of the eyes so that a record of any condition which may exist can be quickly made. There is plenty of room for recording tests made by all instruments of precision and those made by the trial lenses. There are diagrams of the ear and nasopharynx and provision made so that all tests usually practiced may be quickly recorded. In the diagnosis of diseases most of the affections met with are printed with a square at the left, so that only a cross has to be made in it to indicate the diagnosis. On the left-hand page space is provided for the name, age, occupation, residence, single, married, widowed; date of the visit, condition of the patient, treatment, and of amount of charges for the visit. Stubs are provided for pasting in everything connected with the case, such as letters with replies, perimeter charts, etc.; so that all pertaining to the case may be seen in one opening of the case book. For an index, Burr's is used.

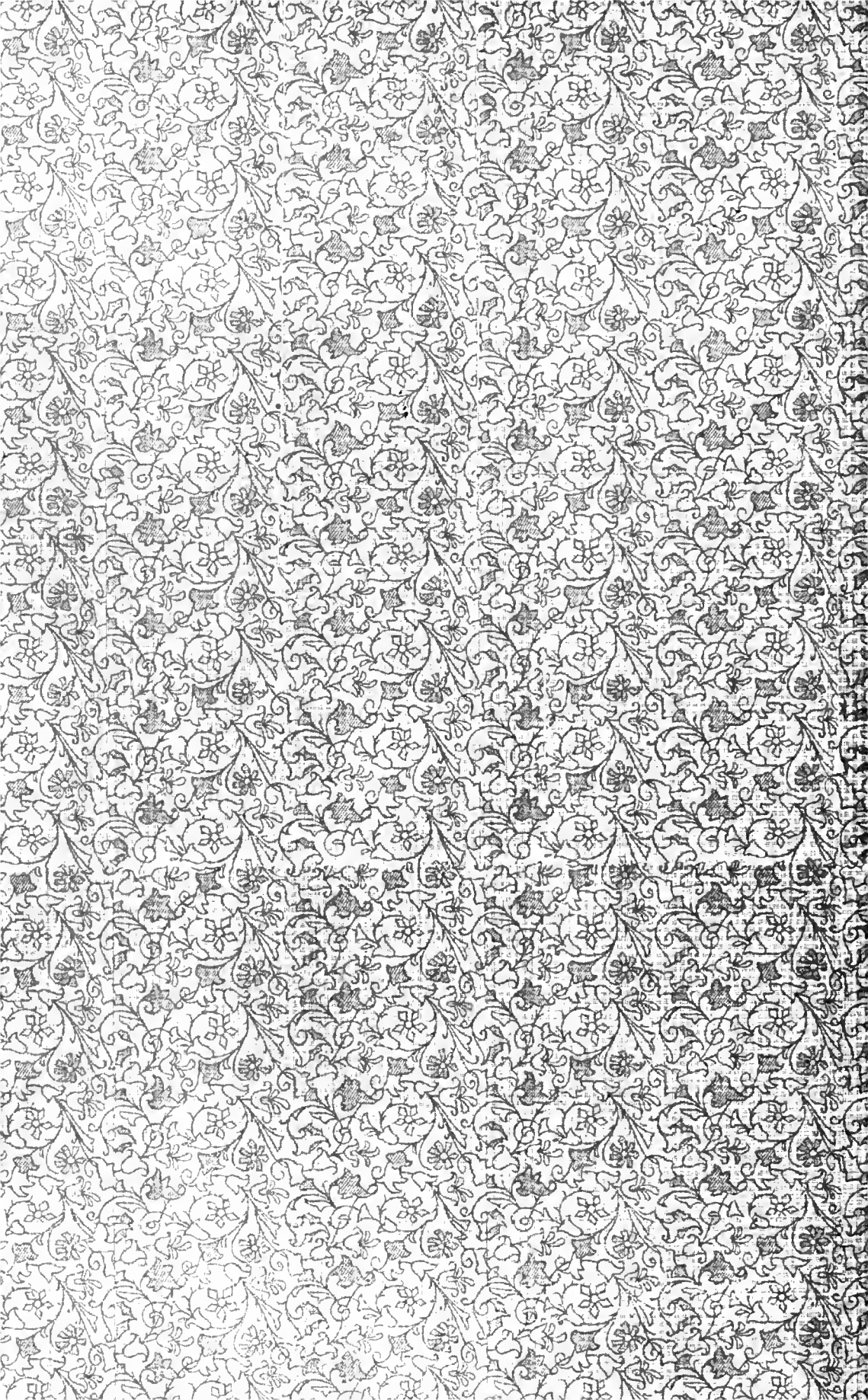
Anyone desiring a sample may communicate with the makers: Loring, Short & Harmon, 474 Congress Street, Portland, Me.

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